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X.

A CONTRIBUTION TO THE BACTERIOLOGY OF THE SOCALLED COCCOBACILLUS FETIDUS OZENÆ (PEREZ), WITH ADDITIONAL NOTES ON THE TREATMENT OF CLINICAL OZENA BY MEANS OF POLYVALENT VACCINES MADE FROM THE SAME ORGANISM.*†

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GENERAL INTRODUCTION.

At the sixty-sixth annual session of the American Medical Association, held in San Francisco, June, 1915, one of us (Horn) presented a preliminary report on "The Etiology and Treatment of Ozena."[148]

The work up to this time had been done at the laboratories of Stanford University and the St. Francis Hospital. Some months later, Dr. Ernst Victors, director of laboratories, San

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Francisco Polyclinic and Postgraduate College, was invited to join in the investigation, and the following study is the result of work done in his private laboratory. We take this opportunity of thanking Stanford University and the University of California for the generous way in which they have placed at our disposal all of the ozena material of these two institutions; and to the many physicians in various parts of the United States who have sent material or referred cases to us for observation and treatment.

We believe that the contention of Perez,[122] made thirteen years ago, that the coccobacillus fetidus ozenæ is the true and only cause of ozena, has not been successfully controverted. Our bacteriologic work has consisted in the more exact study of the morphologic characteristics of this organism; and an endeavor to correlate it with the bacillus bronchisepticus. Its assumed close relationship to the Friedlander group seems by our work to be fully settled in the negative. We believe that it has absolutely nothing to do morphologically with the bacillus of Abel or other members of the Friedlander family. That these two organisms exist side by side in the nose, there is no question. In fact, one can observe a typical Perez bacillus and the typical bacillus of Abel and Löwenberg on the same slide; and we have often noted that as the case improved clinically, the Friedlander group takes the place of the Perez bacillus and seems the last to clear up. Since the publication of our preliminary report but one article has appeared which has added any new light to the subject. The article by Burckhardt and Oppikofer[143] advances a theory which we were able to disprove many months ago—viz., that the Perez bacillus was closely related to the Friedlander group. These investigators were almost on the verge of what we consider the crux of our present paper when they casually mentioned in a footnote that one strain of the Perez bacillus furnished them by Hofer was motile. This interesting observation they credited to a contamination. The fact of the motility of this organism is now proved beyond question. Every strain of the Perez bacillus, twenty-nine cases, shows distinct motility, and flagellæ can be easily stained in all cases. The positive complement fixation reactions with strains of the bacillus bronchisepticus, isolated by Prof. Carl Meyers, University of California, puts it indisputably into the class with these organisms.

The work of Torrey and Rahe[142] satisfactorily proved the cause of distemper in dogs, and has been accepted without question by us as a basis for our investigation. It has been frequently suggested, and carefully worked out by Perez,[130] that the infection in ozena probably originates from the handling of dogs. Hofer[118] has added to his polyvalent vaccine a culture from the nose of healthy dogs, although, as far as a study of the literature goes, he does not specifically mention the bacillus bronchisepticus, nor refer to the work of Torrey and Rahe. We are now engaged in a more extensive study of this phase of the subject, but results are not ready for publication.

A review of the work of Perez, the investigators who followed him, and the more recent work of Hofer, need not be repeated here. A résumé in English by Guggenheim,[147] our own preliminary report,[148] and the article by Burckhardt and Oppikofer[143] in German, fully cover the historical phase of the subject.

It might be interesting to restate the questions which were asked in our preliminary report and see whether, in the light of our present work, the questions have been answered.

We are extremely sorry for the pessimistic and somewhat suspicious tone which pervaded our former publication, and which unfortunately was shared by others who saw the work in Vienna, for our recent work has convinced us that the solution of the etiology of ozena is almost at hand, and that very satisfactory results can be hoped for in the treatment of this disease by means of vaccine.

1. "Is the Perez bacillus the true cause of ozena?" As stated elsewhere, taking into consideration our own additions to the morphology of this organism, we consider this question has been answered in the affirmative.

2. "Has not the undoubted cure of certain cases with vaccines made from the Perez organism and the undoubted failure to cure in other cases, clinically similar, weakened our concept of the clinical entity of clinical ozena?" We believe that for the present it is convenient to divide clinical ozena into a Friedlander group and a Perez group, according to the bacteriologic findings, until such a time as we can more accurately determine what clinical relation the Friedlander group bears to the coccobacillus, or what its rôle is in the etiology of the disease. Our detailed statement of this problem in the

bacteriologic part of the paper, shows that we believe that ozena is in reality a clinical entity.

3. "Is it not possible that cases of ozena, clinically identical, may be cured by several different vaccines?" When this question was asked, we had in mind the improvement which some cases seemed to show after the administration of stock mixed vaccine containing staphylococci, streptococci, micrococcus catarrhalis, etc. Our recent work has convinced us that these saprophytes clear up as readily with the pure Perez vaccine as they did with the old mixed stock vaccines.

4. The last question as to whether we have been able to produce a successful vaccine we can answer decidedly in the affirmative. A study of our case histories would seem to substantiate this contention. We attribute our apparent failures, spoken of in the latter part of the preliminary report, to be due to the fact that we had not, up to that time, prepared a proper vaccine.

ANALYSIS OF THE CASE HISTORIES.

As will be seen by reference to Charts 1 to 5, seventy-one cases have been studied. Chart 1 shows that in fifteen cases of simple atrophic rhinitis, neither the Perez bacillus nor the bacillus of Abel was once found. This simply goes to show that these two organisms are not present in this type of case, nor are crusts and odor usually present, and also shows that the bacillus is never found in the normal nose. This last point has also been confirmed in a large series of routine laboratory examinations, the details of which are not given here, and although the Abel type is never present, other members of the Friedlander group are frequently found in accessory sinus and other infections.

This leaves forty cases (Charts 2—4) in which a diagnosis of clinical ozena was made, either by ourselves or by others. We think a great deal of reliance can be placed on these clinical diagnoses, as they were all either examined by us, sent from the University clinics, or the histories accompanying the cases were conclusive. In twenty-eight of these forty-eight cases the Perez organism was shown to be present, or fifty-eight per cent. The best previous results, given by Hofer and others, have been about thirty per cent, possibly indicating an improvement in our methods of isolation.

A most important fact seems to us to be that in the remain-

ing forty-two per cent of the cases only the Abel bacillus or members of the Friedlander group were found. In other words, in every case where a clinical diagnosis of ozena was made, we have been able to demonstrate one or the other of these two groups as the predominating organisms.

These findings were not always obtained at the first bacteriologic examination. Often as many as fifteen examinations would be necessary before we could convince ourselves of the presence of the Perez organism. In one case (M. H., Chart 2) the bacillus pyocyaneus was found in pure culture nine times; but finally, by various methods of cultivation, we were able to demonstrate the Perez bacillus.

In fact, we have about come to the conclusion that in every case of true clinical ozena, one of these two organisms or both of them must be found, otherwise the technic is at fault. We are inclined to the belief that investigations now being made will enable us to explain why the Perez organism cannot be demonstrated in every case, and that further improvement of technic will increase the proportion of the Perez to the Friedlander group.

It will be noted that in the thirty-two cases given in Chart 2, four are given as staphylococci. In these cases the crusts were sent from distant points, such as South Dakota, Idaho, etc., and were from three to six days in the mail. The appearance of the crusts was typical of ozena, but a strong overpowering stench of sulphuretted hydrogen was present, showing that contamination had taken place, and giving the probable reason for our inability to demonstrate the Perez bacillus.

AGE AND NATIONALITY.

The ages range from nine to fifty years, showing that our cases had neither to do with the very young nor the very old. Of the thirty-two Perez cases (Chart 2) sixteen were males, fifteen were females, showing the proportion to be about equal. Fifteen of the cases were Americans, the rest being of various other nationalities, showing that in America at least the proportion of ozena among the native born is almost as large as among the foreigners. The various percentages on these points among the Friedlander ozenas (Chart 3) is approximately the same.

CASES OF SYPHILITIC OZENA.

An interesting sidelight has been thrown on the matter of syphilitic ozena (Chart 4). Through the kindness of the University of California, eight cases of active syphilis, where the clinical diagnosis of syphilitic ozena was made, were referred to me for bacteriologic study. In three of these cases the Perez bacillus was found. In the other cases, merely staphylococci. In the cases where the Perez was not found, the crusts and odor, although marked, did not in any way resemble the typical odor of the Perez organism. We consider that the odor which accompanies these cases is usually due to the destructive process of the syphilis, and not to a specific organism. These three cases only go to prove that a true ozena can coexist with active syphilis, and, as far as at present can be seen, is not favorably influenced by the most vigorous antisyphilitic treatment. The fact that the Perez bacillus was found in the congenital, tertiary, and active secondary stages shows that it is not necessarily confined to any one stage.

Whether the depraved state of the mucous membranes found in syphilis and tuberculosis predispose to infection with the Perez bacillus is a point now under study and will be reported on later.

METHODS OF EXAMINATION.

In order to get a proper perspective of the good done by the treatment, the suggestion, which has been made by previous investigators—viz., to allow the patients to continue their own treatment at home—has ordinarily been followed out. If the patients were in the habit of washing the nose once or twice a day, this was ordered continued until such a time as it was considered unnecessary by them.

Inasmuch as the majority of these patients have lost their sense of smell, the amount of odor present can be best determined by the examiner. Very little weight is given to the statement of the parents, because after about the third injection the odor disappears to such an extent that they are unable to detect it by ordinary methods.

In a marked case of ozena the odor can be distinctly perceived at from four to six feet and even more. When in the charts the expression "No odor" is used, it means that if when the examiner places his face in immediate proximity

to that of the patient, after several exhalations through the nose no trace of odor can be perceived, the case is considered odor free. If, at this contact examination, a very slight but still perceptible odor is present, the case is labeled "Slight odor." In the nearly cured cases it is necessary to repeat this experiment many times, moving the face back and forth in front of the patient, in order to detect any odor, because under certain conditions the odor is not perceived for several minutes or until five or six breaths have been exhaled through the nose by the patient.

In the apparently cured cases the disagreeable odor is absolutely gone. The breath has not even the heavy smell which often accompanies chronic catarrh, but is more the character of the breath of young and healthy infants. It is necessary to note here that we imagine that we can determine a slight difference between the odor of a Friedlander ozena and a Perez ozena. The latter has a peculiar, sweetish, nauseating, lightish odor; the former, an overpowering, heavy stench. We doubt whether any scientific value can be placed on this observation, but it is interesting in the light of future work.

It seems important to distinguish between a bacteriologic and a clinical cure. As yet, except in but one or two cases, we have not been able to convince ourselves that any of the cases are bacteriologically cured. In some of the cases where the clinical improvement has been greatest, we have been unable to find the Perez bacillus. In other cases, after the Perez bacillus has disappeared, the Friedlander bacillus seems to have taken its place.

In those cases which have been several months without treatment of any kind, where the crusts and odor have entirely disappeared, where the appetite and weight have increased, and where the general statements of the patient are that they feel immensely improved and relieved, we have adopted, for the sake of convenience, the term "clinically cured." Naturally, these cases will be kept under observation for a period of at least a year before any more definite statement can be made. It must be distinctly understood that we have never claimed a case as finally cured.

Because of the expense entailed, the Wassermann examination was not demanded in every case. This subject has already been very thoroughly gone into by various authors, and

we believe, with them, that syphilis plays absolutely no rôle in the etiology of ozena.

No extensive discussion will be entered into with regard to the rôle which accessory sinus disease plays in the cause of ozena. Our impression is that in practically every case the mucous membranes of the antrum and sphenoid are infected with the Perez organism. We have been able to prove in one case at least that a pure culture could be obtained from the antrum, and in many cases have been able to obtain the Perez bacillus in pure culture from the mucous membrane of the sphenoid.

That the entire mucous membrane of the nose, including the submucous tissue, and all of the accessory sinuses are involved, is further substantiated by the fact, that even in those cases where cultures from the nasal mucous membrane showed only the various saprophytic organisms, it was extremely easy to get the Perez bacillus in pure culture from the submucous tissues overlying the turbinates.

This method is as follows: The turbinate was first cocainized, then painted with pure tincture of iodin. A heavy needle, attached to a Luer syringe, was punctured through the iodin area and the needle withdrawn and a bouillon tube was inoculated. The tube always remained sterile and showed that no bacteria were carried into the submucous tissue by the needle. The needle was again punctured through the iodin area, the point moved about, near or below the periosteum of the turbinate, until a slight hemorrhage had taken place, and then the plunger partially withdrawn. The few drops of blood were placed in bouillon, and usually, in the genuine ozena cases, a pure culture of the Perez bacillus was obtained. These experiments were repeated on both the inferior and middle turbinate. We have not proved the extension to the septum and other parts of the nose, but we have every reason to believe that in a well marked case it is to be found in practically all of the mucous membranes of the nose and accessory sinuses. It would seem a far cry to attribute to the accessory sinuses a primary etiologic rôle. It seems to us far more logical to assume that the infection is an extraneous one, and spreads by contiguity from the mucous membranes of the septum and turbinates.

Although in this series of cases, because of insufficient time,

meagerness of the material, and the fact that we did not wish to complicate the picture of the cure by anything other than straight vaccin therapy, the sinuses were not drained, yet we feel, that in order to produce the best results, it is absolutely essential, in all cases, to drain the sinuses whenever there is a suspicion of trouble. In fact, we believe that it would be a good plan in every case, at the beginning of the treatment, to do several punctures of the antrum with thorough irrigation. If, as is often the case, on account of the atrophy, the sphenoids can be entered, it would be advisable to thoroughly wash out these cavities. In several cases where the odor did not properly disappear, we found a retention of pus in the sphenoid or antrum, and a cleaning out of the same, would rapidly result in an odor free nose.

The striking results we have obtained by vaccine therapy in ozena is unquestioned (Chart 5), and having established this fact, we should use every possible accessory aid in the elimination of pus foci. We are convinced that no amount of vaccine treatment will ever cure a case of Perez infection in the retained pus of an antrum or a sphenoid.

METHODS OF VACCINE ADMINISTRATION.

We have found that to obtain the best results it is necessary to work out the proper interval of dose for each individual patient. To do this expeditiously, we begin every case with 200 million initial dose, and have the patient report in twenty-four hours. If no symptoms indicating reaction are reported, we give 400 million, and have the patient report again in twenty-four hours. If there are no symptoms of reaction, we give 800 million, and continue to have the patient report every twenty-four hours, doubling the dose (every twenty-four hours) until the patient reports definite symptoms of reaction (headache, general malaise, poor appetite, temperature and occasionally nausea). The dose causing this reaction we consider the minimum therapeutic dose. Usually by the time the dose is 800 million the patient reports reaction symptoms. At each injection we then have the patient report in seventy-two hours, when we give the next dose. We increase the minimum therapeutic dose by 200 or 400 million, according to the symptoms reported by the patient. The symptoms indicating non-increase of dosage or extension of the seventy-two-hour inter-

val to ninety-six hours or more, are fatigue, depression, loss of appetite, subnormal temperature, and failure of pulse to return to rate at time of previous injection. We believe the three and four day interval will be found to give better results. Improvement should be unquestionable after the fourth dose following the dose producing reaction, and the first course of injections should number about twenty, at the above (three or four-day) interval.

In our experimental work we have used many different combinations of polyvalent Perez vaccines. It was only toward the last of our work that we were enabled by means of agglutination experiments to produce vaccines which are fairly satisfactory. In the last series we have used only vaccines whose potency has been increased by passage through animals. We have a plan in our new experiments for greatly increasing the efficiency of the stock vaccine. Hofer[130] speaks of his bitter disappointment in finding some of his results negative because of improperly prepared vaccine; and we feel that if the present series of cases could have been treated with the vaccine which we are now using, our own results would have been far more satisfactory. For these reasons some of the apparent contradictions are explained, and some of the long series of injections could have been greatly shortened.

The dosage of four to five billion seems unnecessarily large, and as the vaccine becomes more potent the number of bacteria per cubic centimeter will be greatly lessened. Even in small children we have never seen any ill effects with large doses, and we must strongly urge that in order to get the best results the dose must be pushed to the extreme limit of reaction. It is remarkable how quickly the patients, after having one severe reaction, become almost immune to subsequent doses, no matter how large.

CASE HISTORIES.

Case 1.—J. M., nine years; male; Mexican.

Family History.—Mother suffers from rhinitis atrophica fetida, Perez type. One brother same.

Past History.—Bothered since three years of age. Frontal headaches. Marked odor.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis. Greenish crusts anterior end of middle turbinate. Odor not particularly marked.

September 22, 1914. Right side blocked with greenish crusts. Odor not marked.

October 21, 1914. Odor extremely offensive. Crusts abundant. Injection, Sherman's No. 36, 3 million.

October 28, 1914. Crusts better. Odor better. Injection, 6 million Sherman's No. 36.

December 5, 1914. Crusts decidedly better. Odor marked. Reaction slight.

December 8, 1914. Injection, 6 million No. 36.

December 19, 1914. Odor and crusts about the same as at first.

December 26, 1914. Injection, 8 million No. 36.

January 9, 1915. Injection, 7 million No. 36.

January 23, 1915. Following last injection marked local reaction. Odor much improved. Injection, 5 million No. 36.

January 30, 1915. Injection, 6 million No. 36. Some improvement.

April 10, 1915. Crusts somewhat improved. Odor still strong. Beginning stock Perez vaccine No. 1. 150 million.

April 15, 1915. Slight local reaction. No headache. 250 million.

April 17, 1915. Second day following last injection, odor markedly increased. Crusts loosened up. Odor entirely disappeared. No crusts. Injection, 300 million.

April 24, 1915. Odor still present, but not so severe as formerly. Crusts diminished. Injection, 500 million.

May 1, 1915. Considerable reaction from last injection. Crusts very much decreased. No odor. Mother marks improvement.

May 8, 1915. No crusts. No odor. Injection, 500 million.

May 15, 1915. General condition somewhat improved. Injection, 500 million.

May 22, 1915. Condition much better. Injection, 1 billion.

June 5, 1915. General condition much improved. Odor better.

June 19, 1915. No crusts. Slight odor.

March 28, 1916. Patient has been away nearly a year. It is possible to get a slight suspicion of odor if the child breathes

directly in your face. There are no crusts in the nose. Mother says general condition about the same. Injection, 500 million No. 6 stock Perez. Mother says odor is decidedly improved since first treatment, but if allowed to go without washing, odor returns.

March 29, 1916. Decided local reaction. Nose not washed for twenty-four hours. Decided odor.

March 31, 1916. No crusts. No odor. Injection, 1 billion stock No. 6.

April 5, 1916. Slight reaction. No crusts. No odor. Injection, 1 billion stock No. 6.

April 8, 1916. Injection, 1½ billion. No crusts. No odor.

Note.—This was one of the early cases in which we tried stock mixed vaccines to clear up the saprophytes. The treatment at first seemed to benefit, but later the odor and crusts returned. When the Perez was started, April 15th, it was noted that after the second injection the odor became decidedly worse. This is an observation which we have made in many subsequent cases. Seven injections were given, when the case remained away nearly a year. On return the condition was markedly improved over the first examination, but the Perez was still present. The case was started with a new series of Perez vaccins.

Case 2.—H. M., aged eight years; male; Mexican; schoolboy.

Family History.—Mother suffers from rhinitis atrophica fetida. Perez type. One brother same. (J. M., Case 1.)

Past History.—Nose trouble for three years. Strong odor at all times. Crusts worse on right side.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis. More marked on left than right. Inferior turbinate almost disappeared. Left nostril completely blocked with greenish, foul-smelling crusts. Odor very offensive. No crusts on right side.

November 21, 1914, to January 29, 1915, four injections of Sherman's No. 36.

April 13, 1915. Injection Perez No. 1, 200 million. Since last injection of No. 36, crust formation is about the same. Odor about the same.

April 17, 1915. Severe reaction from injection. Pale and

nauseated. Absolutely no odor or secretion in the nose. No crusts or pus.

April 24, 1915. Injection stock, 300 million. No odor. General condition improved. No crusts.

May 1, 1915. Condition of the nose about the same. No odor. Strong reaction. Following last injection, headache for two days. Nausea and malaise.

May 8, 1915. Injection stock, 250 million. No crusts or odor in nose.

May 15, 1915. Injection stock, 250 million. No reaction. Nose the same. Hg., eighty-five per cent.

May 22, 1915. Condition about the same. Feeling of malaise following last injection.

June 5, 1915. General physical condition improved. Odor less.

June 19, 1915. No crusts or odor in nose.

March 28, 1916. Injection, stock No. 6, 300 million. Case not seen for nearly a year. Patient has been very well since last examination. No trouble with nose. Mother has not noticed odor. No odor. Left side more crusts.

March 29, 1916. Nose not washed for twenty-four hours. No crusts. No odor. Decided local reaction from last injection.

March 31, 1916. Injection stock No. 6, 1 billion. No crusts. Slight odor.

April 5, 1916. Injection stock No. 6, 1 billion. Severe general reaction from last injection. Headache, malaise and fever lasting forty-eight hours.

April 8, 1916. Injection stock No. 6, 1½ billion. Slight reaction from last injection. No crusts or odor.

Note.—The same observation which we made on J. M., his brother, applies here. H. M. always took the treatments badly. The reactions were very severe, keeping him in bed for several days at a time. The same dose on his brother, but one year older, had but very little effect. Case still under treatment.

Case 3.—B. N., aged thirteen years; female; American; schoolgirl. First examination, July 26, 1915.

Family History.—Negative. Sister complains of odor. Odor very offensive at times. Not characteristic at time of examination.

Past History.—Has had odor and crusts in nose for years.

Bacteriologic Examination.—Perez.

Examination.—Typical ozena, with crusts covering inferior turbinate, both sides. Middle turbinate, right, more atrophic than left. Marked pharyngitis. Process extends down almost to larynx. Larynx not involved. Odor marked.

July 26, 1915. Injection stock Perez, 750 million.

August 6, 1915. Injection, 1 billion. Patient says odor is better. No crusts. Scanty amount of secretion. Absolutely no odor. Slight general and local reaction.

August 16, 1915. Injection, 500 million. Crusts smaller. No odor.

August 21, 1915. Injection No. 36, 2 million, No. 38, 3 million. Examination of crusts gives Friedlander. Reaction from last Perez vaccine was marked; headache and fever. Local reaction.

August 28, 1915. Injection No. 36, 8 million. Increase in crusts. No odor or headache. Sister noticed odor yesterday.

August 30, 1915. Injection Perez, 400 million. Condition in nose much improved. No odor. Light reaction.

September 4, 1915. Injection, 400 million. Condition about the same as in last examination. Crusts much easier to blow out.

September 13, 1915. Injection, 500 million. Considerable crusts in nose; more left than right. No odor.

September 18, 1915. Injection, 500 million. Condition much improved. No odor and no reaction.

September 25, 1915. Injection, 1 billion. Slight headache following injection. Feels about the same generally. Absolutely no odor. Monday following last injection, noticed a decided odor.

October 2, 1915. Injection, 1 billion. Very little odor. Crusts very slight and easy to remove. Right sphenoid opened.

October 9, 1915. Injection, 1 billion.

October 18, 1915. Right antrum washed; probe puncture. Pus very thick; almost impossible to wash out.

Note.—This case received about twelve injections of the earlier vaccines. The child was very stubborn, hard to handle, and the father suspicious of the vaccine treatment. The parents noted the improvement, which, at the last examination, gave slight odor; crusts slight and easy to remove. The pa-

tient objected to the treatment to the right antrum, and did not return for further injections.

Case 4.—J. P., aged twenty years; female; American.

Family History.—Negative.

Past History.—When ten years of age, noticed odor, with great amount of crusts. Crusts very hard to remove. Gradually growing worse. Various types of treatment, with apparently no benefit. No operations. Wassermann negative.

Bacteriologic Examination.—Pure Perez.

Examination.—Marked high grade atrophic rhinitis. Typical ozena of nasopharynx. Saddle nose. Nauseating odor.

Treatment—September 8, 1915. Injection, 1 billion stock Perez.

October 9, 1915. Reaction general; chills, headache and fever.

October 15, 1915. Injection, 1 billion. Patient has not douched nose for one day. Very few crusts. No odor.

October 18, 1915. Injection, 2 billion. Increase of odor. No crusts.

October 25, 1915. Injection, 2 billion. No odor or crusts.

November 3, 1915. Injection, 2 billion. No odor or crusts. Gained five pounds.

November 12, 1915. Injection, 250 million autogenous.

November 18, 1915. Injection, 1 billion autogenous. Nose not washed for forty-eight hours. No odor or crusts.

December 1, 1915. Injection, 1 billion autogenous. Gained three pounds. No crusts or odor.

December 9, 1915. Injection, 1 billion autogenous. Nose not washed for one day. Decided odor, but no crusts. Patient says that she has had more or less odor.

December 13, 1915. Injection stock No. 3, 1 billion. Strong odor, as she had at beginning of treatment. Very few crusts.

December 21, 1915. Injection stock vaccine No. 3, 1 billion. Odor remains slight but marked.

December 28, 1915. Injection stock No. 3, 1 billion. No odor or crusts.

January 4, 1916. Injection stock No. 3, 1 billion. Nose washed daily. Very slight odor and few crusts.

January 10, 1916. Injection autogenous No. 2, 500 million. Slight odor and crusts.

January 14, 1916. Injection autogenous No. 2, 1 billion. No crusts or odor.

January 18, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts.

January 25, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts.

February 1, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts.

February 4, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts. Has not washed nose for two days. Does not have headaches any more. Patient in every way greatly improved.

Note.—This is the most brilliant result obtained in our series. When first seen, the girl was almost septic; the bridge had sunken, she was afflicted with a profound melancholia, and had very little appetite. The odor was terrible, and one was unable to stay in the same room with her. She had received various forms of treatment ever since she was a child. When examined, April 15th, or two months after last injection, she had gained nineteen pounds, her complexion was rosy, her disposition was happy, and her appetite had returned. There has been absolutely no odor or crusting during this time, and she considers herself entirely well. No further treatments will be given until some of the old symptoms are complained of.

Case 5.—W. S., aged twenty-eight years; male; Russian Jew; machinist.

Family History—Unknown.

Past History.—Has complained for years of stinking catarrh. Does not know how long. History of playing with dogs unknown. Notices himself that the odor is gradually growing worse. Says it is especially bad in wet weather.

Bacteriologic Examination.—Perez.

Examination.—Typical ozena fetida. Odor at four feet. Extreme atrophy. Few scattered whitish crusts over the whole of nose. Wassermann negative.

Treatment.—March 9, 1916. Injection stock Perez No. 6, 400 million.

March 10, 1916. General reaction. Odor decidedly worse. Loss of appetite, headache and tired feeling. No injection.

March 11, 1916. Injection stock No. 6, 600 million. Odor

still present, but decidedly less. Crusts much improved. Small crusts on middle turbinate.

March 13, 1916. Injection No. 6 stock, 1 billion. Few crusts. Odor barely perceptible.

March 15, 1916. Injection No. 6 stock, 2 billion. Examination of cultures show Perez positive. No crusts or odor. Slight amount of pus in region of ethmoids.

March 17, 1916. Injection No. 6 stock, 3 billion. Odor strong. Nose perfectly clean, except for one crust on the anterior end of middle turbinate. No reaction.

March 20, 1916. Severe general reaction from last injection, which lasted up to today. Loss of appetite, headache and fever. No local reaction.

March 28, 1916. Injection No. 6 stock, 4 billion. Absolutely no crusts. Slight suspicion of odor. Patient for the first time noticed his own odor.

March 29, 1916. No crusts, slight odor; no reaction.

March 31, 1916. Injection stock No. 6, 3 billion. Absolutely no odor or crusts. Mucous membranes bright red in color and general feeling greatly improved.

April 5, 1916. Injection No. 6 stock, 4 billion. Condition same.

April 8, 1916. Injection stock No. 6, 5 billion. No reaction. No crusts, but slight odor.

Note.—This is one of the most interesting cases under treatment. The reactions were always marked, the odor was worse after the second injection and entirely cleared up after the third. In this case, for the first time we have been able to convince ourselves that the mucous membranes of the nose change from a decided atrophic condition to normal looking. Several careful observers who saw this case April 14, 1916, were unable to diagnose an atrophic condition, but considered the mucous membranes almost normal. One of the striking features of this case is the tremendous increase in the general wellbeing of the patient.

Case 6.—J. S., aged thirty-five years; male; American.

Family History.—Negative.

Past History.—Gives no history of trouble in nose. Decided huskiness of voice for last twelve years.

Bacteriologic Examination.—Perez.

Examination.—Larynx has small node on the right cord. The interarytenoid space distinctly thickened, always covered with slimy secretion and crusts. It is very difficult to make out whether or not there is an active ulceration. Both cords red and inflamed, and thickened and covered with a shiny crust. Marked atrophic rhinitis nonfetida. Very little crust over middle turbinate; not characteristic of ozena. Wassermann negative.

Treatment.—February 18, 1916. Injection stock No. 5, 100 million.

February 19, 1916. Injection stock No. 5, 100 million. No reaction.

February 21, 1916. Injection stock No. 5, 1 billion. For the first time since treatment, crust has disappeared from the anterior commissure of the larynx. Mucous membrane of the larynx more moist and healthy looking.

February 23, 1916. Injection stock No. 5, 1 billion. No reaction. Voice and larynx decidedly cleared. No crusts in interarytenoid space. No crusts in nose. Last examination of culture shows only staphylococcus.

February 25, 1916. General reaction, with malaise. Local reaction. No crusts in interarytenoid space. Larynx mucous membrane decidedly more pinkish; all signs of congestion lost.

February 26, 1916. Injection stock No. 5, 1 billion.

Note.—This case was certainly not one of clinical ozena. The stubbornness with which the larynx condition responded to treatment led us to examine the nasal secretion, where the Perez was found in pure culture. The five injections of Perez vaccine had a marked effect on clearing up the laryngeal condition. From an examination of the larynx, we would not have suspected a laryngeal ozena, and examination from the secretion of the larynx never showed the Perez organism.

Case 7.—S. P., aged twenty-seven years; female; German.

Family History.—Negative.

Past History.—When fifteen years of age first noticed disagreeable odor in nose. When twenty years old noticed headaches, from which she has suffered ever since.

April, 1910, curetted anterior ethmoids on right side. Following this, in June and October, intranasal operations on right side. February 4th, double Killian operation. Stinking

pus from right ethmoid, frontal and sphenoid. Three weeks after operation left sphenoid opened.

Bacteriologic Examination.—August 15, 1915. Pure Perez.

April 6, 1912. Left sphenoid and ethmoid cells curetted as widely as possible.

April 26, 1912. Autogenous vaccine made from sphenoid shows Friedlander in pure culture. Injection autogenous vaccine, 10 million. From August 26, 1912, to October 1, 1912, twenty injections of autogenous vaccines, with varying success. Stopped treatment for two years. On return, condition same. Started another course of ten injections of autogenous vaccines without benefit, finshing March 23, 1915.

April 29, 1912. Injection autogenous, 20 million.

April 5, 1915. Injection mixed Perez, 500 million. No crusts, no odor.

April 19, 1915. Injection stock, 2½ billion. Nose much improved.

April 26, 1915. Injection stock, 4 billion. No reaction.

May 6, 1915. Injection, 4 billion stock. No reaction.

May 12, 1915. Injection stock, 4 billion. No reaction.

May 18, 1915. Injection stock, 4 billion. No reaction.

May 25, 1915. Injection stock, 4 billion. No reaction. Patient says she feels better now than she has for years.

July 17, 1915. Injection stock, 750 million.

July 24, 1915. Injection stock, 1 billion. Very slight reaction.

August 14, 1915. Injection stock, 250 million and one-half cubic centimeter of No. 36.

August 18, 1915. Injection stock, 1 billion. No reaction.

August 25, 1915. Injection stock, 1 billion. No reaction.

September 1, 1915. Injection stock, 1 billion. No reaction.

September 15, 1915. Injection stock, 1 billion. No reaction.

September 22, 1915. Injection stock, 1 billion. No reaction.

September 29, 1915. Injection stock, 1 billion. No reaction. No crusts or odor. Washes nose every day.

October 6, 1915. Injection stock, 1 billion. No reaction.

October 15, 1915. Injection stock, 1 billion. No reaction. Patient douches nose every other day.

October 20, 1915. Injection stock, 2 billion. No crusts in nose. No odor.

November 10, 1915. Injection stock, 2 billion. Patient had a severe reaction following last injection.

Note.—This case has been under treatment since April, 1912. She had an extensive double pansinuitis. Innumerable intranasal operations, including double Killian, were done for the relief of the discharge and headaches. It was not until August, 1915, that we suspected a Perez infection. Up to that time she had had many autogenous vaccines given, apparently with little benefit. The case cleared up wonderfully with the Perez vaccine, and has not been seen for several months. The physical improvement in this patient is perfectly remarkable, but all the credit cannot be given to the Perez vaccin. It is a type where the combined surgical and vaccine treatment gave the best results.

Case 8.—C. W., aged twenty-nine years; male; American; dentist.

Family History.—Negative.

Past History.—Has had typical ozena odor since nineteen years of age. No improvement, in spite of various vaccine treatments. Condition about the same for last few years. Denies lues. Strong sweetish odor on breath. Loss of smell.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophy of both sides. Whitish crust over opening of sphenoids. Process more extensive on left side. Marked sweetish odor.

Treatment.—December 24, 1915. Injection No. 3 stock Perez, 250 million.

January 20, 1915. Injection autogenous Perez, 500 million. No reaction.

January 28, 1916. Injection autogenous, 500 million. No reaction.

February 4, 1916. Injection autogenous, 1 billion. No reaction.

February 12, 1916. Injection autogenous, 1 billion. No reaction.

February 19, 1916. Injection autogenous, 1 billion. No reaction.

February 27, 1916. Injection autogenous, 1 billion. No reaction.

February 29, 1916. Patient says he noticed a decided improvement in crusts. Has gained three pounds. Friends have

not noticed odor. Examination: Right side perfectly dry and clean. Mucous membranes fairly moist. Left side no sign of crusts. Small amount of mucopus in back of throat. Absolutely no odor, even when patient breathes directly into face. Probably some involvement of the left sphenoid.

March 3, 1916. Bacteriologic examination of secretion shows Perez bacillus no longer present.

March 4, 1916. Injections of autogenous vaccine as follows: March 4, 1½ billion. March 8, 1916, 2 billion; marked reaction; slight local, but a general tired and malaise feeling. March 11, 1916, 2 billion; more reaction than with the last injection.

March 13, 1916. Patient previous to treatment with vaccines had both frontals, sphenoids, ethmoids, and left antrum opened intranasally. Nose perfectly clean, with the exception of a small crust in the region of the left sphenoid. Culture taken from left sphenoid. Very slight, but barely perceptible, odor when directly in contact with the face, and then only at times. There is undoubtedly more or less infection in left sphenoid. General condition very much improved, appetite good, weight about the same. Patient is able to work on his patients with every degree of comfort. No one has mentioned anything about odor. Left sphenoid reopened. No sign of pus in same.

March 28, 1916. Injection No. 6 stock, 2½ billion. Slight secretion in region of left sphenoid. Absolutely no crusts or odor. Dr. Waiss present at this examination, and also detects no odor.

March 15, 18 and 22, 1916. Injections of No. 6 stock, 2 billion.

Note.—This is probably one of the two (vide, J. P.) most brilliant clinical cures in our series. The patient, a dentist, had been obliged to give up his professional work, and had been treated by various forms of vaccine therapy, but without any benefit. At the last examination he had gained six pounds. There is absolutely no trace of odor on his breath, his general condition is markedly better, and he has received no treatment of any kind for over two months. He considers himself cured. Treatment will be omitted until some of the old symptoms develop.

Case 9.—A. J., aged thirty-four years; male; Russian; tailor.

Family History.—Negative.

Past History.—Since twelve years of age, foul smelling ozena, gradually getting worse. Several intranasal operations without benefit. Sprays nose every night. Weight, one hundred and forty-four pounds. Odor strong but not characteristic of Perez; possibly Friedlander. Has lost sense of smell. Wassermann negative.

Bacteriologic Examination.—Staphylococci.

Examination.—Inferior turbinate and middle turbinate hard to make out. Mucous membrane very thin and covered with greenish crusts. Odor perceptible at three feet.

Treatment.—February 16, 1916. Injection No. 3 Perez stock, 100 million.

February 17, 1916. Injection No. 3 stock, 400 million. No reaction.

February 18, 1916. Injection No. 3 stock, 500 million. No reaction.

February 19, 1916. Injection No. 3 stock, 600 million. Slight reaction from last injection. If patient breathes directly into face, one gets odor.

February 21, 1916. Injection No. 5 stock, 500 million. Slight suspicion of odor when in direct contact with nose. Crusts practically disappeared, except in region of the sphenoid.

February 23, 1916. Injection stock No. 5, 400 million. Odor entirely disappeared. One small crust on the anterior end of the middle turbinate. No reaction.

February 25, 1916. Injection No. 5 stock, 500 million. No reaction from last injection. No crusts or odor.

Note.—This was a typical clinical ozena, but the Perez or the Abel bacillus was never demonstrated. Seven injections gave marked improvement, but the case did not return for treatment.

Case 10.—A. M., aged twenty-one years; female; housewife.

Family History.—Negative.

Past History.—Stinking catarrh for last eight years. Played with dogs as a child. Not improved with treatment. Washes nose once a day and sprays nose once a day.

Bacteriologic Examination.—Perez.

Examination.—Extremely extensive atrophic rhinitis, with whitish crust on the anterior turbinate, and foul smelling pus. Odor perceptible at three feet.

Treatment.—March 9, 1916. Injection stock vaccine No. 6, 200 million.

March 10, 1916. Patient declares herself decidedly improved after first injection. Head feels clear, nose not so stopped. Crusts about the same. Odor is decidedly improved. Injection, 400 million stock No. 6.

March 11, 1916. Injection stock No. 6, 600 million. Shows slight general reaction. No odor or crusts.

March 13, 1916. Absolutely no odor. A few whitish crusts on anterior end of middle turbinate. Had a severe headache for a couple of hours on March 11th, from injection. Injection stock No. 6, 1 billion.

Note.—After four injections the improvement was simply wonderful, but unfortunately the case did not return for further treatment. Patient expressed herself as cured.

Case 11.—B. A., aged twenty years; male; American.

Family History.—Negative.

Past History.—Since fourteen years of age has complained of crusts and odor. People around him say that the odor is very disagreeable. Patient says he had very heavy crusts. The patient has had a regular obsession regarding his odor. In his profession as priest he found it necessary to douche his nose six to eight times a day, using several quarts of water at a time, and then was unable to satisfactorily get rid of the crusts.

Bacteriologic Examination.—December 1, 1915, Perez. January 6, 1916, Perez. February 19, 1916, Friedlander and staphylococcus aureus. February 22, 1916, pure Friedlander.

February 23, 1916. No odor, few crusts on anterior end of middle turbinate. Patient is now taking another course of stock vaccine No. 5.

Treatment.—Has had about twelve to fifteen injections. No injections since December 15, 1915. Has not washed nose for forty-eight hours. Absolutely no odor. Small whitish crusts.

Note.—The present condition of the patient is a marvelous change from his former unhappy condition. He is able to conduct his professional work without the fear of disgusting the people he comes in contact with, and as a matter of habit

douches his nose but once a day. Since last December the treatment has been irregular, and he still receives, mainly for experimental purposes, an injection about every fourteen days.

Case 12.—J. A., aged twenty-two years; male; Russian Jew. Referred by Dr. G. Richards, Fall River, Mass.

Family History.—Mother has ozena.

Past History.—For four years atrophic rhinitis, with odor. Odor very offensive. Crusts very thick and hard to blow out. Douches nose every day.

Bacteriologic Examination.—November 29, 1915, Perez.

Examination.—High grade atrophic rhinitis. No crusts or odor.

Treatment.—November 29, 1915. Injection stock Perez, 500 million. Nose not washed for three days. No typical ozena crusts. Strong offensive odor.

December 3, 1915. Injection stock, 1 billion. Nose not washed for four days. Characteristic odor, few crusts. Patient has not noticed odor since last treatment. No reaction.

December 14, 1915. Injection stock No. 3, 1 billion. No crusts or odor.

December 24, 1915. Injection stock No. 3, 1 billion.

December 30, 1915. Perez positive.

January 3, 1916. Injection stock No. 3, 1 billion. Nose not washed for twenty-four hours. No crusts or odor.

January 10, 1916. Injection stock No. 3, 1 billion.

January 13, 1916. Perez positive.

January 17, 1916. Injection stock No. 3, 1 billion. No reaction.

January 24, 1916. Injection stock No. 3, 1 billion. No crusts or odor. Gaining in weight and appetite. Feels decidedly better.

February 1, 1916. Injection stock No. 3, 1 billion. No reaction.

February 8, 1916. Injection stock No. 3, 1 billion. Nose not washed for nine days. No odor, very slight crusts.

February 19, 1916. Absolutely no odor or crusts. Nose washed once in two weeks, although not necessary.

March 4, 1916. No crusts or odor. Nose not washed for two weeks.

March 13, 1916. Perez positive.

April 3, 1916. No injection since February 8th. No crusts or odor.

Note.—This case is a good example of a clinical but not bacteriologic cure. He is a Perez carrier, and although he has had no injections since February 8th, and no odor has been noted since December 14th, we can at any time demonstrate Perez in pure culture.

Case 13.—H. F., aged thirty-three years; male; French: porter.

Family History.—Unknown.

Past History.—Atrophic rhinitis. Patient complains that crusts and odor are worse during wet weather. Used to complain of headaches, but since he has been washing nose does not notice them. Washes nose with douche once a day.

Bacteriologic Examination.—Perez.

Examination.—October 29, 1915. Atrophic rhinitis both sides. Cultures taken from nose show Perez. When examined had no crusts or odor. Told not to wash nose and report in one month.

November 4, 1915. Injection No. 1 Perez stock, 500 million. Examination: Marked atrophic rhinitis. Few scattered crusts of whitish color on anterior end of middle turbinate and in region of sphenoid. Medium but characteristic odor. Patient has lost entire sense of smell. Patient's friends notice odor.

November 6, 1915. Slight local reaction. Nose same.

November 13, 1915. Injection Sherman's No. 36, 0.6 cubic centimeters.

November 16, 1915. Autogenous vaccine, 250 million. Crusts slight, odor strong. Has not washed nose for three days.

March 11, 1916. Patient returns, saying that condition is the same as before, and is obliged to wash his nose every day, and crusts and odor are as bad as ever. Examination: Condition about the same as in last examination; it is impossible to get but a small crust for examination. The nose is moist, and although patient washed nose this morning, there are no crusts or odor.

March 11, 1916. Injection No. 6 Perez stock, 400 million.

March 13, 1916. Injection No. 6 stock, 800 million. Slight local reaction. Condition about the same.

March 14, 1916. Injection No. 6 stock, 1 billion. Slight local reaction.

March 15, 1916. Injection, No. 6 stock, 2 billion. No crusts or odor.

March 18, 1916. Injection No. 6 stock, 2½ billion. Feels decidedly better, has not washed nose for three days. No crusts or odor. No reaction.

March 20, 1916. Injection No. 6 stock, 3 billion. Patient says he is feeling decidedly improved. No crusts or odor. No reaction.

Note.—This case has been treated for years in various clinics. The odor and crusts were slight at all times, and although he showed a pure Perez and admitted improvement under treatment, he fails to return for his injections.

Case 14.—M. G., aged twenty-one years; female.

Family History.—Negative.

Past History.—Never played with dogs. Parents noticed odor and crusts from age of six years. Gradually grew worse. Several intranasal operations; no benefit. Sherman's No. 36, long course, very little benefit. June, 1915, to August 26, 1915, ten injections of Perez mixed stock, with marked improvement of all symptoms and odor. Headaches have entirely disappeared and have not returned. People around her noticed same improvement. Before taking vaccine it was necessary to go and have crusts removed twice a week, and odor was unbearable. For last four weeks crust have not been removed from nose. Crusts are now so loose that she can douche them out. This could not be done before injections. Douches nose t. i. d.

Examination.—Tremendous improvement so far as crust is concerned. Typical atrophy. Slight whitish crust middle turbinate and septum. Slight odor.

Treatment.—August 26, 1915. Stock Perez, 500 million.

September 2, 1915. No odor. Pus from sphenoid both sides. Injection of 500 million.

September 11, 1915. Condition same. Injection of 500 million.

September 18, 1915. Slight reaction. 500 million injection. Slight crust. No odor.

September 27, 1915. Injection of 1 billion.

September 28, 1915. Right sphenoid opened.

October 4, 1915. Injection of 1 billion.

October 11, 1915. Injection of 1 billion.

October 19, 1915. Injection of 2 billion. Crusts in region of sphenoid.

October 26, 1915. Injection of 2 billion. No odor; slight crust.

October 27, 1915. Severe reaction from injection.

November 3, 1915. Injection of 2 billion.

November 13, 1915. Injection of 2 billion.

November 24, 1915. Injection of autogenous, 250 million. Examination: Crusts in region of sphenoid. Patient unable to wash out crust. Slight odor.

December 3, 1915. Injection of autogenous, 500 million.

December 13, 1915. Sterile antrum puncture gives pure Perez. Nasal secretion pure Perez.

December 21, 1915. No crusts or odor. Appetite good. Gained four pounds. Secretion in region of sphenoid. Injection of autogenous, 1 billion.

December 27, 1915. No odor or crust. Crust easier to remove. Injection of No. 3 stock, 1 billion.

Note.—The patient received in all fifteen injections of various Perez vaccines. The condition at the beginning of the treatment was extremely unsatisfactory, the patient having had a great deal of operative work in the nose and was extremely hysteric. Her blood coagulation point was low, so that twice following work on the sphenoid the nose bled for nearly a week. When last seen there was no odor, no crusts, weight and appetite greatly improved, and general nervous condition better. The patient asked to be allowed to omit treatment for a period of six months. In this case we were able to show both in the antrum and sphenoid a pure culture of the Perez.

Case 15.—M. H., aged twenty-one years; female; American; stenographer.

Family History.—Negative.

Past History.—Odor and crusts since childhood. In 1910 crusts became much worse. Played and kissed dogs as a child.

Bacteriologic Examination.—Pyocyaneus. Six examinations from July 15, 1915, to December 4, 1915. December 4, 1915, Perez positive.

Examination.—Marked atrophic rhinitis. No crusts. Slight amount of pus.

Treatment.—August 13, 1915. Injection stock No. 1, 500 million.

August 21, 1915. Injection No. 36, 3 million; No. 38, 3 million.

August 24, 1915. Injection No. 36, 5 million; No. 38, 5 million.

August 28, 1915. Injection No. 38, 8 million; No. 36, 8 million. Condition very much improved. No crusts or odor.

August 30, 1915. Injection No. 2 stock, 600 million.

September 3, 1915. Slight reaction. Headache, fever. Slight local reaction. Crusts easier to blow out. No odor.

September 6, 1915. Injection No. 2 stock, 600 million. Considerable reaction from last injection.

September 18, 1915. Injection No. 2 stock, 1 billion. Condition much improved.

September 25, 1915. Injection No. 2 stock, 1 billion. Patient has feeling of lightness and well-being. Mucous membrane very dry.

October 2, 1915. Condition about the same. Injection of 1 billion.

October 4, 1915. No reaction. Considerable crusts on right side.

October 9, 1915. Injection of 1 billion. No crusts, no odor and no headaches.

October 16, 1915. Injection of 1 billion.

October 23, 1915. Injection of 2 billion No. 2 stock. Nose in very good shape.

October 30, 1915. Injection of 2 billion. No odor and no crusts.

November 6, 1915. Injection of 2 billion. Very little crusts. No odor.

November 13, 1915. No odor, no crusts and no headaches. Has not washed nose for one week. Injection of 2 billion.

November 20, 1915. No crusts. No odor. Instructed to not wash nose for one week.

November 29, 1915. Blood, crusts and submucous cultures taken. No crusts or odor. Last examination by Dr. Victors and myself shows still no Perez organism.

December 6, 1915. Injection of 1 billion stock No. 3. No crusts or odor.

December 13, 1915. Patient says she had considerable odor

all week. Douched the nose today. No crusts and no odor. Injection of 1 billion No. 3 stock.

December 20, 1915. Slight reaction. Slight dizziness, nausea and headache. No odor. Injection stock No. 3, 1 billion.

January 5, 1916. Patient has not washed nose since last visit. No odor, very few crusts. Patient is losing weight and appetite. Injection stock No. 3, 1 billion.

January 19, 1916. Injection No. 3 stock, 1 billion. Patient feels extremely well two days following injection.

January 27, 1916. Condition same. Injection stock No. 3, 1 billion.

February 4, 1916. No odor or crusts. Patient discharged for one month.

Note.—In this case nine examinations were made before the Perez could be demonstrated. There was always a very abundant growth of bacillus pyocyaneus. Stock vaccines were tried, to clear up the pyocyaneus infection, but without benefit, and it was not until a Perez vaccine was used that the case improved until she was discharged clinically cured. In all, eighteen injections were given. In this case the submucous examination showed the pyocyaneus.

Case 16.—H. K., aged twenty-five years; female; Irish.

Family History.—Negative.

Past History.—Had odor and discharge since childhood. Odor has not improved, but seems to be getting worse. Did not play with dogs. Odor extremely offensive, crusts entirely blocking nose.

Bacteriologic Examination.—July, 1915, Perez. September 22, 1915, Perez. February 14, 1916, Friedlander and Perez on same plate.

Treatment.—From July to November about twenty injections by Dr. Victors. When the case was examined by me she was free from odor and crusts, and was generally very much improved and satisfied. The case is still receiving injections from time to time for experimental purposes.

Case 17.—A. L., aged thirty-five years; male; German. First eamination, April 8, 1916.

Family History.—Negative.

Past History.—The day case appeared for examination no crusts or odor. No diagnosis of ozena could be made other than marked atrophic rhinitis.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis.

Treatment.—April 8, 1916. Injection stock No. 6, 500 million.

April 10, 1916. Injection stock No. 6, 1 billion. Slight local reaction.

Note.—April 5, 1916. When seen today, reports fairly satisfied. Feels generally much improved, has no odor or crusts. Case still under treatment.

Case 18.—C. M., aged thirty-nine years; female; Mexican; housewife.

Family History.—Negative.

Past History.—Dry catarrh. Crusts always right side. Formerly strong odor, but none at present.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis. Inferior turbinate almost disappeared; also the middle turbinate. Covered with dark greenish crusts. Odor slight but characteristic.

Treatment.—March 31, 1916. Injection stock No. 6, 500 million.

April 5, 1916. Injection stock No. 6, 1 billion. Local reaction. No odor or crusts.

April 8, 1916. Injection stock No. 6, 1½ billion. Slight reaction. Condition about the same.

Note.—This woman brought her two sons to the clinic. (Cases 1 and 2.) An examination of the mother, as above given, gave a pure Perez. The woman did not complain of any nasal trouble. Case still under treatment.

Case 19.—R. P., aged seven years; female; Italian; schoolgirl.

Family History.—Negative.

Past History.—Strong foul odor all her life.

Bacteriologic Examination.—Friedlander.

Examination.—Marked atrophic rhinitis. The entire nose is filled with crusts. Crusts are blackish, very foul smelling. Odor is perceptable at four to six feet.

Treatment.—March 24, 1916. Injection stock Perez No. 6, 500 million.

March 25, 1916. Injection No. 6 stock, 800 million. No reaction.

March 27, 1916. Injection No. 6 stock, 1 billion. Odor and crusts decidedly better. No reaction.

March 30, 1916. Injection stock No. 6, 1 billion. Neighbors say odor is better.

March 31, 1916. Injection stock No. 6, 1½ billion. No reaction.

April 4, 1916. Injection stock No. 6, 1½ billion. Slight odor and crusts.

April 8, 1916. Injection No. 6 stock, 2 billion. No reaction. Condition same.

Note.—This case shows the large Friedlander bacillus, and, like, the V. M. case, is extremely unsatisfactory in treatment, although marked improvement is noted.

Case 20.—L. P., aged twenty-eight years; male; Italian; fisherman.

Family History.—Negative.

Past History.—From age of six years complained of small crusts and odor, with headaches.

Bacteriologic Examination.—Friedlander.

Examination.—Medium atrophic rhinitis. Absolutely no crusts. Slight but characteristic odor of ozena. Nose not typical of ozena. Never washes nose. Typical large black crusts in the nasopharynx, which are very difficult to remove.

Treatment.—February 19, 1916. Condition unchanged. Odor scarcely perceptible. Marked glazed look to the nasopharynx. Larynx dry but not typical of ozena. Injection stock Perez No. 5, 500 million.

February 21, 1916. Injection No. 5 stock, 500 million. No reaction.

February 26, 1916. Injection No. 5 stock, 500 million. No reaction.

February 29, 1916. Injection No. 5 stock, 800 million. Crusts about the same. No odor. Slight local reaction.

March 1, 1916. Injection No. 5 stock, 1 billion. Crusts and odor about the same.

March 4, 1916. Injection No. 5 stock, 2 billion. Slight local reaction.

March 6, 1916. No crusts or odor. Nasopharynx same. General reaction without headache. Patient complains of weakness in knees and loss of appetite. No injection.

March 9, 1916. Injection No. 6 stock, 2 billion. No crusts or odor.

March 14, 1916. Injection No. 6 stock, 2 billion. Nasopharynx beginning to show a slight degree of moisture, otherwise condition the same.

Note.—This case has not reported for treatment for over a month. At last examination there seemed to be a slight improvement in the pharyngeal condition. The odor, which was always very slight, did not return.

Case 21.—A. D., aged thirty years; female; Italian.

Family History.—Negative.

Past History.—Has complained of this disease since childhood; gradually grown worse. Did not play with dogs as a child. Patient has retained smell. Smells her own odor. Odor characteristic of Friedlander.

Bacteriologic Examination.—Friedlander.

Examination.—Marked atrophic rhinitis. Crusts on left middle turbinate. None right.

Treatment.—February 24, 1916. Injection Perez stock No. 5, 100 million.

February 25, 1916. Injection No. 5 stock, 200 million. Crusts about the same. Odor possibly slightly improved. Patient says she feels better. Had slight local reaction.

February 28, 1916. Injection No. 5 stock, 500 million. Slight local reaction.

March 1, 1916. Injection No. 5 stock, 1½ billion. Crusts much drier. No free pus. Very slight odor noticed only by approaching near to nose.

March 3, 1916. Injection No. 5 stock, 2 billion. Slight local reaction. No headache. Crusts decidedly improved. Slight but faint odor.

March 6, 1916. Distinct general reaction from last injection. Pain in arm, general malaise and loss of appetite. No injection.

March 9, 1916. Injection No. 6 stock, 2 billion. Very few crusts. Very slight but typical odor.

March 13, 1916. No injection. Odor practically gone. On account of patient's general reaction, injections stopped for nine days.

March 24, 1916. Injection No. 6 stock, 2 billion. One large crust on left side without odor; right side entirely clean. Slight odor.

March 30, 1916. Injection No. 6 stock, 2 billion. Slight odor. Few crusts.

April 3, 1916. Injection No. 6 stock, 3 billion. Slight odor, few crusts.

April 10, 1916. Injection No. 6 stock, 3½ billion. No crusts, slight odor.

Note.—This is what we call the septic type. The color is bad. She takes her injections badly, and, as is often the case with this form of infection, the result is slow and the improvement not nearly so satisfactory as in the Perez infections.

Case 22.—E. D., aged thirteen years; male; American; schoolboy.

Family History.—Unknown.

Past History.—The odor first noticed on breath three years ago, and has gradually grown worse. Has large amount of crusts in the nose, which are very difficult to remove. Odor very offensive. For the last six months has been decidedly worse.

Bacteriologic Examination.—Friedlander.

Examination.—Hypertrophy of both sides. Nose entirely filled with large crusts. Odor is extremely offensive; perceptible at five feet. The odor is that of a Friedlander more than a Perez.

Treatment.—March 28, 1916. Injection stock Perez No. 6, 500 million.

March 29, 1916. Injection No. 6 stock, 1 billion. Decided local reaction.

March 31, 1916. Injection No. 6 stock, 1 billion. Strong odor. Nose entirely blocked with crusts.

Note.—Case still under treatment.

Case 23.—M. Z., aged thirty-two years; female; Hungarian.

Family History.—Negative.

Past History.—At eighteen years of age noticed odor and crusts. Sense of smell and taste lost.

Bacteriologic Examination.—Perez.

Examination.—Saddle nose. High grade atrophic rhinitis. Impossible to make out turbinates. Entire nose covered with dark crusts. Odor extremely offensive.

Treatment.—March 12, 1915. Injection stock, 100 million.

April 7, 1915. Injection stock, 250 million.

April 14, 1915. Injection stock, 250 million. Odor decidedly better; crusts about the same.

Note.—This case, an ignorant Hungarian woman, expressed herself as believing that vaccine treatment was a form of poison and refused further injections.

Case 24.—E. O., aged eight years; male; Mexican; schoolboy.

Family History.—Mother and one brother show Abel.

Past History.—Very large crusts in nose, which are hard to remove. Very foul odor, but more typical of the Friedlander type.

Bacteriologic Examination.—Abel bacillus.

Examination.—Nose completely blocked with large black crusts, which are extremely hard to remove. They are very hard and dry.

Treatment.—January 8, 1916. Injection stock Perez, 100 million.

January 22, 1916. Injection stock, 200 million. No reaction.

January 27, 1916. Injection stock No. 3, 500 million.

January 29, 1916. Injection autogenous vaccine, 250 million. No reaction from last injection. Crusts are more moist and easier to remove.

February 1, 1916. Injection autogenous vaccine, 500 million. Crusts the same; no odor.

February 5, 1916. Odor and crusts worse. Injection autogenous vaccine, 1 billion.

February 15, 1916. Injection autogenous vaccine, 1 billion. No odor; crusts decidedly improved. No reaction from last injection.

Note.—This is the typical bacillus mucosus capsulatus of the large plump type. The treatment, both in this case and his brother and mother, both of whom had exactly the same infection, has been very unsatisfactory. The cases have been very irregular in attendance. The use of the autogenous vaccines in this case was an experiment of little value. Patient since above dates has been taking Perez stock vaccines, with marked improvement.

Case 25.—M. C., aged fourteen years; female; Chinese; schoolgirl.

Family History.—Negative.

Past History.—Very indefinite.

Bacteriologic Examination.—Friedlander.

Examination.—Odor very strong and perceptible at three feet. Marked atrophic rhinitis. Crusts entirely blocking the nose.

Treatment.—March 16, 1916. Injection Perez stock No. 6, 200 million.

March 17, 1916. Injection No. 6 stock, 400 million. Condition about the same.

March 18, 1916. Injection No. 6 stock, 1 billion. Nose entirely free from crusts. No odor. No reaction.

March 20, 1916. Injection No. 6 stock, 1½ billion. No crusts or odor.

March 24, 1916. Injection No. 6 stock, 2 billion. No crusts or odor. No reaction.

March 28, 1916. Injection No. 6 stock, 3 billion. Condition same.

March 31, 1916. Injection No. 6 stock, 3 billion. Nose perfectly free from crusts and odor. No reaction.

April 5, 1916. Injection No. 6 stock, 1 billion. No crusts or odor. No reaction.

April 8, 1916. Injection No. 6 stock, 3 billion. No crusts or odor. No reaction.

Note.—This case is one of the most brilliant improvements in the Abel group. The crusts cleared up quickly and remained so. The odor has not returned since the third injection. Case under treatment.

Case 26.—W. B., aged twenty-seven years; female; English.

Family History.—Negative.

Past History.—When seven years of age noticed trouble. Has treated more or less; no improvement. Used to play with dogs. Has lost sense of smell.

Bacteriologic Examination.—Friedlander.

Examination.—Typical atrophic rhinitis of true ozena. It is impossible to make out the turbinates. Crusts plaster the entire inside of nose. Odor foul and heavy. Wassermann negative.

Treatment.—February 25, 1916. Injection No. 5 Perez stock, 200 million.

February 26, 1916. Injection No. 5 stock, 400 million. No reaction.

February 28, 1916. Injection No. 5 stock, 500 million. Crusts and odor about the same. No reaction.

March 3, 1916. Injection No. 5 stock, 1 billion. Patient feels decidedly better.

March 6, 1916. Injection No. 5 stock, 2 billion. No headaches, crusts or odor.

March 10, 1916. Injection No. 5 stock, 2 billion. General reaction. Malaise.

March 15, 1916. Injection No. 5 stock, 2½ billion. Slight reaction. No crusts or odor.

March 20, 1916. Injection No. 5 stock, 2 billion. Severe reaction one hour after injection. Nausea, headache, lasting two days. Nose feels better. No odor. One large crust on left size.

March 24, 1916. Injection No. 6 stock, 2 billion.

March 27, 1916. Injection No. 6 stock, 3 billion.

April 5, 1916. Injection No. 6 stock, 3 billion. Slight whitish crusts on both sides. Slight odor on breathing directly into face. No reaction.

April 8, 1916. Injection No. 6 stock, 4 billion. No crusts or odor.

Note.—This case has been under treatment for years in various clinics. She has received many injections of various types of vaccines. There is an infection of the left sphenoid and ethmoid with causes constant secretion and crusting in the left side. The injections have given the patient more satisfaction than any previous treatment, and the odor is certainly decidedly better.

Case 27.—V. M., aged thirteen years; female; American; schoolgirl.

Family History.—Negative.

Past History.—Mother noticed ozena trouble since child was four years old. Last three or four years odor remains the same. Odor worse at times.

Bacteriologic Examination.—Friedlander, Abel.

Examination.—Marked atrophic rhinitis with thin white crusts spreading over turbinate, septum and floor of nose. Nose washed twice a day.

Treatment.—November 23, 1915. Injection Perez stock, 250 million. Nose not washed for one week. Slight ozena odor. Right nostril entirely blocked with blackish crusts. Left filled with whitish crusts. Mother says that odor is not as bad now as it gets to be.

November 26, 1915. General reaction, headache, nausea and dizziness. Local reaction. Crusts and odor less.

December 8, 1915. Injection stock, 250 million. Nose not washed for one day. Large typical crust on left side; very slight characteristic odor of ozena. Patient says that odor is improving. Crusts very hard to remove. Patient generally looks very much improved.

December 15, 1915. Injection stock No. 3, 500 million. Condition about the same.

December 29, 1915. Injection stock No. 3, 250 million. General reaction immediately following last injection. Nausea, headache, chills, fever and dizzy. Reaction lasted three days. Patient noticed that within two days from injection crusts practically disappeared, and they are much easier to blow out. Examination: Absolutely no odor; few crusts far back.

January 5, 1916. Injection Sherman's No. 36. Entire nose filled with crusts. Condition about the same as in first examination. No reaction. Odor strong, but not typical of ozena.

January 8, 1916. Injection autogenous vaccine, 300 million. Slight odor. No crusts.

January 10, 1916. Injection autogenous vaccine, 600 million. Slight nose bleed from last injection. Slight odor. Crusts easier to remove. No reaction.

January 15, 1916. Injection autogenous vaccine, 600 million. No crusts or odor. No reaction.

January 21, 1916. Injection autogenous vaccine, 1 billion. Condition same. No reaction.

January 26, 1916. Injection autogenous vaccine, 1 billion. Odor same. Few crusts. Patient notices great improvement.

January 31, 1916. Injection autogenous vaccine, 1 billion. Crusts much easier to remove. No odor. Has not washed nose for twenty-four hours. No reaction.

February 4, 1916. Has not washed nose for two days. No odor; few crusts. Treatment stopped for two weeks. Local reaction.

February 10, 1916. No odor; slight amount of crusts.

February 19, 1916. Injection No. 5 stock, 100 million. Crusts decidedly improved. No odor.

February 23, 1916. Injection No. 5 stock, 200 million. Nose not washed for one day. Interior plastered with whitish crusts. No odor. In spite of presence of the crusts, patient does not notice them, and they are not so difficult to remove.

Note.—This case was placed in the Friedlander group. The improvement while under treatment was remarkable, but often in three or four days the nose would fill up as noted on January 5th, and it would seem that no improvement had taken place. The patient was very stubborn and hard to handle, and finally refused further treatment.

RABBIT EXPERIMENTS.

The pathologic changes in the nose of rabbits, following the intravenous injections of cultures of the Perez bacillus, have been carefully investigated by Hofer[113] and more recently by Burckhardt and Oppikofer[143]. The local manifestations in the turbinates following one or more injections are marked. Our specimens show the whole gamut of changes from the intense congestion following a fatal dose to a complete atrophy with actual destruction of the bone.* The observations of Burckhardt and Oppikofer were carefully and painstakingly made, but their negative results and findings are so at variance with our own and the findings of Hofer and Perez, that we can only attribute them to the use of cultures so often subcultured that their virulence had been greatly lowered.

Our interest in the matter, however, has been chiefly in methods of producing rapid agglutinating sera, determination of toxicity, and fatal dose. It is not our purpose to criticise the paper of Burckhardt and Oppikofer. Our rabbit protocols are not repeated here in detail. (See Chart No. 6.) We do, however, feel that working with but two subcultures furnished by Hofer, their results are bound to be unsatisfactory and inconclusive. Our work, as shown in Chart No. 6, included fifteen strains. The most unsatisfactory results were obtained with cultures furnished by Hofer. These were marked originally I and II, but whether they were the same cultures furnished Burckhardt and Oppikofer, also marked I and II, we are unable to state. It was only after animal passage that we were able to obtain satisfactory results with these strains.

*Details of histopathologic studies have been purposely omitted and will be presented in our forthcoming paper.

As a rough basis for the estimation of the dose, to a fresh vigorous twenty-four hour agar transfer, five cubic centimeters of salt solution was added, and an emulsion made, as shown in Chart No. 6. The rabbits injected with the killed cultures, following the method of Hofer, lost flesh very rapidly, and it seemed impossible to secure an agglutinating serum. A dose of one and one-half to two cubic centimeters (Chart No. 6, Rabbits Nos. 1 to 10) seemed invariably fatal. The changes in the nose were marked, but we could not isolate the Perez from the nasal secretion or use the sera for agglutinating purposes.

As soon as live cultures were used (Nos. 11 to 49), the rabbits held their weight better, an agglutinating serum was easy to obtain, and some of the rabbits lived as long as three months. One animal lived nearly six months, but the average length of life is two to three months, after three to five injections of a maximum dose of two cubic centimeters.

We have recently found that if the dose is started very small and repeated at frequent intervals, a serum agglutinating as high as 1 to 2,000 can be obtained in a few weeks. (Nos. 38 to 43.) It is well to try the agglutinating power after the fourth injection, and if present to keep the rabbit without further injection, or allow at least a ten day interval to elapse. In this way, if all conditions are favorable, the animals can be kept alive for six months.

Experiments are now under way to determine the endo- and exo-toxin content, and to try and explain the great variability in the toxicity of the various strains. A study of Chart No. 6 will prove useful in determining the maximum dosage. The details are self-explanatory. Full autopsy protocols are not given here, as they shed no light on the question of dosage.

We cannot understand the inability of Burckhardt and Oppikofer to demonstrate the Perez bacillus in the nasal secretion of injected rabbits. In spite of most careful search, they failed in every case.

As shown in Chart No. 6, the organism was recovered, in many cases, in pure culture and proved by agglutination. The typical coryza came on after the first injection, and in two cases, small healthy rabbits, previously shown to be free from Perez, contracted acute coryza by cage contact with the dis-

eased animals, and the Perez was demonstrated in pure culture from their nasal secretions.

We were also able to demonstrate the pure culture from the heart, blood and lungs, the latter habitat being especially significant in the light of our observations concerning the relation of the Perez bacillus to the bacillus bronchisepticus of dogs.

The statement that they were never able to produce atrophy of the turbinates is directly at variance with our own experience. Our specimens show many such cases. Their claim that the virulence of the culture was proved because the animals died of acute septicemia with large doses, is open to serious objection. The animals probably died from acute toxemia which follows the administration of large doses.

Their contention, that the absence of crusts and odor in the nose of rabbits is an important point against the specificity of the Perez infection, is unconvincing. Crusts, per se, have nothing to do with ozena. The formation of crusts depends on the chemical composition of the secretion. The odor is always less marked when the secretion acquires a watery character. In our own rabbit experiments, where ten to fifteen animals were confined in one cage, a very characteristic odor could be noted ten to fifteen feet from the cage. In certain animals where the process was far advanced, a characteristic ozena odor was noted as soon as the head was split open. Their admission, that they never saw atrophy in their animals, is the best proof that they were not dealing with advanced cases.

BACTERIOLOGIC STUDIES.

This part of the paper embraces a more intensive biologic study of "coccobacillus fœtidus ozænæ" (Perez) than contained in the previous description of Hofer[113], and comparative studies of Bacillus bronchisepticus and Bacillus mucosus capsulatus. In some instances we are at variance with Hofer concerning the morphologic and metabolic behavior of the organism. Bacillus bronchisepticus, exhaustively studied by Ferry[140], McGowan[141], Torrey and Rahe[142], and determined by them as being the specific organism of canine distemper, morphologically almost identical and biologically in many instances similar to the Perez organism, and the advanced hypothesis that the infection of fetid ozena is carried by dogs, presents an

interesting complex. The frequency with which organisms of the Friedlander group are isolated from typical and atypical ozena cases, biologic comparatives, changes clinical and of the nasal bacterial flora under immunologic measures with bacillus Perez vaccin, led us for the time being to the consideration of a possible bacterial mutation. Burckhardt and Oppikofer[148] have placed the organism tentatively among the Friedlander-aerogenes group. By our own and other studies we are inclined to doubt this contention, and tend rather to place the organism with the colityphoid group. Some bacteriologists may be inclined to place the organism with the hemorrhagic septicemia group.

Morphology.—The vegetative cell from twenty-four-hour agar cultures falls into one of two groups: (1) A distinct coccoid rod with a poorer staining protoplasmic equator, though not sufficient to confuse as a diplococcus. The ends are round. Dimensions from 0.75—1.25×0.4 microns. (2) A larger and thicker bacillus which stains more uniformly. Ends less rounded. Dimensions, 1.0—1.5×0.4—0.6 microns. A relative constancy in the sizes of young culture organisms is encountered; pleomorphism, as described by Hofer, appearing only in old cultures. The bacillus from young broth cultures has no distinction from cultures from solid media. In old broth cultures the organism is larger and pleomorphism is marked. Chain formation, except for occasionally two or three elements, never occurs.* The bacillus is uniformly Gram negative. Stains with ordinary dyes, though not intensely. Bipolar intensity in coccoid type. Spores or capsule are never developed.

Agar Plate Colonies.—In twenty-four hours at 37° C., surface plate cultures are plainly visible. Round, slightly elevated colonies, one to two millimeters in diameter. Edge entire. Translucent and lustrous by reflected light, bluish by transmitted light. In forty-eight hours colonies are doubled in size, circular or slightly ameboid, slightly elevated, flat or slightly convex. Edge undulate. The colony becomes slightly less transparent and somewhat whitish. Microscopically the colony is of high refractile index and internal structure homo-

*Long chain formation with bacillus bronchisepticus is the rule; in young broth cultures appearing in hanging, drop-like, long slender rods.

geneous, later becoming finely granular; center grumose in old colonies. Old colonies show little variation except for increased size to about eight millimeters in diameter, central opalescence and lessening of luster. Blueness by transmitted light of the less dense periphery prevails.

Agar Stroke.—In twenty-four hours at 37° C., a luxuriant growth has developed along line of stroke. Flat, with transparency increasing toward edges. Edge slightly elevated, lobate-lobulate, microscopically showing marginal colony development (lappenbildung). In most instances the medium ultimately becomes deeper yellow or brownish in color—one strain producing an oxidization of the medium to an intense brown.

Plain Broth.—In twenty-four hours there is produced a diffused turbidity of the medium. No change for several days, when a granular deposit develops. There is no scum formation. The medium gives off a sickening odor, but is uncharacteristic.

Motility.—In our experiences, the organism from twenty-four-hour broth cultures is definitely motile in all instances: with certain strains a typhoid-like activity is manifest, but usually the hanging drop shows definite but slow motility with marked activity of occasional organisms.* In older broth cultures (seven days), motility persists, though weaker. Long flagella—usually six are demonstrable.

Gelatin Stab.—In forty-eight hours a delicate filiform growth has developed along line of stab. There is nothing characteristic in older cultures. Gelatin is not liquefied.

Potato.—In twenty-four hours at 37° C., a moist yellowish discoloration of the surface of the medium appears. The outline of the growth is not usually definite. With certain strains the growth may be colorless or slightly whitish. There is no unusual discoloration of the potato, or it may be slightly discolored brown. In forty-eight or seventy-two hours the growth becomes more distinct and raised. The surface becomes uneven and coppery in color. Medium discolored.

Litmus (Azolitmin) Milk.—Certain strains show an increased alkalinity or no change in the reaction of the medium,

*Hofer described the organism as being immotile. This same characteristic is ascribed by Burckhardt and Oppikofer, although in only one of three cultures submitted to them by Hofer was the organism found motile, this characteristic prevailing through animal experiments.

while the greater number show a slight reduction of the litmus, and some produce definite acid production with coagulum.* (See Table 7.) Acid production is slow, and rarely occurs before forty-eight or seventy-two hours. Cultures of various ages show some inconstancy in reaction.

Endofuchsin Agar.—In the first twenty-four or forty-eight hours the colonies are apparently nonacid. After this time the centers of the colonies become red, while the periphery continues white.

TABLE No. 7

Carbohydrate Fermentation and Reaction Record of Various Coccobacillus Ozena Fetidus Cultures.

Culture	Glucose		Lactose		Malt.	Mann.	Sacc.	Litmus Milk		
	G%	A%	G%	A%	G%	G%	G%	1 day	2 day	7 day
Adolphus	–		–		–	–	–	–	–	Lilac
Femol	S1		S1		–	–	–	–	–	A+C
Kelleher	12	3.2	–	1.7	5%	10	10	A+C	A+C	A+C
Strum	–		–		–	–	–	–	–	–
Piffle	–		–		–	–	–	A+C	A+C	A+C
Plant	–		–		–	–	–	–	Alk.	Alk.
Weber	–		–		–	–	–	–	Lil.	A
S. L.	–	1.3	–	1.5	–	–	–	–	A+C	A+C
Mrs. Miranda	10	2.6	–	.05 Alk.	S1	10	10	–	Lil.	A
Vienna		2.8		.7				–	Alk.	Alk.
Stefanovich								–	–	Lilac
Spencer	10		–					–	–	–
Bloomenberg	S1	.6		.2 Alk.				–	–	–
18821	S1	1.9		.7 Alk.				Lil.	A+C	A+C
W. B.	12							–	–	Lilac
H. Miranda,	10		–					–	–	–

(Gas and acid readings are after forty-eight hour incubation.)

Carbohydrate Fermentation.—There is nothing consistent in carbohydrate fermenting capabilities. Culture age gives variation. Some cultures are ineffective with all the carbohydrates. Dextrose is most often fermented (bubble to fifteen per cent). Lactose is never effected. Maltose, mannite and saccharose are variably and indifferently fermented. (See Table No. 7.)

*Hofer states litmus milk is unchanged. Burckhardt and Oppikofer state that acidity is occasionally produced, but that milk is never coagulated.

Acid Production in Dextrose and Lactose Broth.—Reaction change in dextrose broth is usually acid. In twenty-four hours the change may be from none to three per cent acidity. In the next twenty-four hours there may be but little change, but the acidity percentage is most frequently reduced. In seven days little or no change in the reaction takes place.

No appreciable change is observed in twenty-four-hour lactose broth cultures. In forty-eight hours the acidity may reach one per cent, but the change to alkalinity is usual. In seven days little or no change from the forty-eight-hour reading is observed. (See Table No. 7.)

Reduction of Nitrates to Nitrites.—Like bacillus bronchisepticus, the Perez organism can be broadly divided into two groups, according to its nitrifying ability. Of the organisms isolated by us they were almost equally divided in this respect.

Anaerobiasis.—Aerobic, but readily facultates to anaerobic. Growth persists in deep and surface anaerobic cultures. In fermentation tubes both arms are at first uniformly turbid, with increasing turbidity in the open arm.

Odor.—We are unable to detect a marked similarity in the nasal odor of ozena patients and cultures, although culture odor is offensive.* It is not dissimilar to certain other bacterial cultures.

The salient morphologic and biochemic characteristics of the Perez bacillus are: A short, Gram negative, motile coccobacillus, showing more intensive polar staining. It is never capsulated, and spores are not borne. Gelatin is not liquefied. Growth is never mucoid. Agar colonies are somewhat colilike. It may or may not reduce nitrates to nitrites. It is not fastidious in the fermentation of carbohydrats or in the production of acid. Growth on potato is fairly characteristic, although not constant. Odor from broth cultures is offensive, but not characteristic. It may simulate certain of the Abel's bacillus of the Friedlander group, and at times render confusion in morphology and certain biologic characteristics. The Abel bacillus, though, can be made to develop capsule and is never motile. With the most painstaking effort we were unable to develop a capsule with the Perez organism. In size, shape, tinctoral reaction and motility the bacillus bronchisepti-

*Hofer has maintained that young cultures, especially broth, have an odor similar to the characteristic odor of ozena in man.

cus and bacillus Perez are almost identical, although the former is slightly heavier. Grouping according to nitrification is permitted of both organisms. Growth upon potato is similar in most instances. Bacillus bronchisepticus tends to grow in chain formation in broth, is almost strictly aerobic, tends to alkali production and splits no carbohydrats. Bacillus Perez generally differs in all these respects. (Table No. 8.)

In passing, we wish to place emphasis upon motility and frequent acid production, as demonstrated by us, in contravention to the observations recorded in previous publications. From a culture brought by one of us from Hofer's laboratory the organism was proven to be definitely motile. Likewise do we find relatively frequent tendency to acid production with certain cultures, although the medium reaction is unchanged in many instances, and occasionally an increased alkalinity develops.

Agglutination Studies.—A potent agglutinating serum was easily produced by immunization of rabbits with living organisms. A high agglutination titre, 1:1000 to 2000, existed for homologous strains, a relatively lower titre existed for allied strains. For group identification of the organism we found that low serum dilutions (1:160) were sufficient for practical purposes. Even in this dilution strains could be coarsely separated. (Table No. 9.) The agglutinability of the organism is always low until after a suitable laboratory life.

Abel's bacillus and others of the Friedlander group were never agglutinable, even in very low dilutions (1:20). Two strains of bacillus bronchisepticus were non-agglutinable with antisera—Rabbit No. 3 (Adolphus)—the only agglutination experiment carried out with this organism; yet this organism proved to be markedly antigenetic in a complement deviation test with this antisera.

Complement Fixation Studies.—Several complement fixation experiments were carried out for bacterial classification by specific protein determinations, using a Perez antiserum and filtered autolysates of selected Perez, Abel and bronchisepticus cultures. The results of these experiments were striking and conclusive. After determining a constant and definite antigenic power of the Perez autolysate with various immune sera, the antigenic power of the Abel and bronchisepticus autolysates with Perez antisera was studied. The Abel antigen was

TABLE NO. 8.

Comparison Between B. Bronchisepticus and B. Perez,

	B. Bronchisepticus	B. Perez
Morphology	Two forms—short coccobacillus and larger type. 0.75—1.25x0.4—0.5 microns 1.00—1.6 x0.4—0.5 "	Same 0.75—1.25x0.4 and 1.00—1.5x0.4—0.6 microns
Staining	Not intense. More at poles, especially coccoid type. Gram negative.	Same
Motility.	Feebly motile.	Same Occasionally active.
Gelatin.	Not liquefied.	Same.
Reduction of Nitrats to Nitrits	Divided into negative and positive group.	Same.
Agar Colony.	Delicate coli-like. Usually not visible in twenty-four hours.	Similar. Usually visible in twenty-four hours.
Broth Cultures.	Turbid—often scum. Long chain formation.	Turbidity. Never scum. Never in chain formation.
Potato.	Yellow, coppery colored growth. Potato discolored brown.	Usually indistinguishable. Occasionally colorless.
Litmus Milk.	Surface alkalinity.	Frequent acid production. Milk may coagulate. May be no change or alkalin.
Carbohydrats.	No ferment or acid.	Dextrose frequently fermented, with production of acid. Lactose and other sugars rarely or slightly effected.

Anaerobiasis.	Aerobic. Slightly anaerobic.	Grows under aerobic or anaerobic conditions, but preferably aerobic.
Rabbit Inoculation.	Dead culture highly toxic. Living culture better tolerated. Bacterial infarcts without suppuration. Agglutinating serum easily established. Wasting of animal.	Same.
Agglutination	Easily by bacillus bronchisepticus antisera. Not by Perez antisera.	Easily agglutinated by Perez antisera. Bacillus bronchisepticus antisera not attempted.
Complement Fixation.	Autolysate as antigen produces complete deviation of complement with bacillus Perez antisera.	Same.

impotent, and no fixation of complement was brought about. The bronchisepticus autolysate, on the other hand, was equally as active as the Perez antigen in complement binding. Complement fixation tests with serum of clinical and bacteriologic ozena cases were always negative.

TABLE No. 9

Agglutination Record for Group Identification of Coccobacillus Ozenae Fetidus Cultures and Coarse Separation of Strains. Group Differentiation of Abel's Bacillus and B. Bronchisepticus.

Culture	Rabbit Antisera							
	No. 1 (Vienna)	No. 23 (Alpert)	No. 5 (Plant)	No. 7 (Kelleher)	No. 3 (Adolphus)	No. 11 (Femol)	No. 25 (18821)	No. 27 (Piffle)
B. Perez,								
Adolphus	+	+	+	+	+	+	+	
Alpert	+	+	+		+		+	
Femol	+	+	+	−	−	+	+	
Kelleher	−	−	−	+	−	−	−	
Strum	+	+	+	+	+	+	+	
Griffin	+	+	+	+	+	+	+	
18821	+	+	+	+	+	−	+	
Piffle	+	+	−	−	−	+	+	+
Mrs. Miranda	+	−	−	+	+	−	+	
Vienna	+	+		+	+	+	+	
Rab. 11 Femol	−		−	+	+	+		
Rab. 8 Vienna	+		+					
Weber	−	−	−	−	−	+	+	
Kelleher B.	+				+			
Plant	+		+					
S. L.	+				+	+		
Stefanovich	+							
Spencer					+			
B. Abel's,								
Knealy	−		−	−	−	−	−	
Grove	−			−	−	−	−	
Tuckey	−		−	−	−	−		
Faber	−		−	−			−	
B. bronchisepticus,								
No. 1					−			
No. 2					−			

Animal Observation.—Immunologic measures with killed cultures were found unsatisfactory, owing to a marked toxicity of killed cultures and the difficulty of procuring an agglutinating serum of sufficient titre, and were early abandoned. No studies were carried out to determine the toxicity of bac-

terial filtrates, but the toxic fraction is apparently very high.

The animals (rabbits) enjoyed a much greater tolerance to living cultures, and the immunity response was marked. Under cultural injections (intravenous) the animals rapidly lost in weight, and ultimately succumbed to intercurrent infections.

The changes in the nose are described elsewhere in this paper.

Under injections of large doses, bacterial infarcts formed in various portions of the body. These did not go on to suppuration. Not infrequently such infarcts developed on the ears, and cultures from these showed viability after many months.

The organism was isolated from the heart blood, nose and lungs. In one instance we were able to isolate the organism from the nose of another animal that had received no injections, but had been caged with an injected animal.

Experimental inoculations by submucous injection were positive in that the organism was recovered with associated atrophic changes.

Clinical Bacteriologic Considerations.—From forty cases of typical ozena under observation the Perez organism was recovered in twenty-eight instances. In not a few instances we were compelled to make repeated attempts before the organism could be isolated. Isolation of the organism is beset with no difficulties except when the accompanying bacterial flora contains weedy overgrowing types. The commonest offenders are the bacillus pyocyaneus and organisms of the Friedlander group.

The Abels bacillus and others of the Friedlander group are deserving of special consideration. Of the forty cases studied by us, some of the Friedlander group, usually Abel's, were isolated in twelve instances to the exclusion of positive Perez findings. In five instances of an initial positive Perez diagnosis Friedlander organisms appeared in cultures after Perez vaccine therapy with negative Perez cultures. In one instance (Kelleher) Abel's and Perez bacilli were easily isolated from the same culture.

Our search for Perez in plates giving colonies of the Abel's bacilli was not as thorough as it should have been, owing to the belief, for a considerable time entertained by us, that a bacterial separation of fetid ozena cases might be made, or

that the organisms were coetiologic. In the light of recent studies we are inclining to the belief that the activities of the Perez bacilli in the nose creates a condition symbiotic for Friedlander organisms, and that negative Perez findings from typical cases were due to technic difficulties. The belief is now entertained by us, though not yet verified, that through the activities of the Perez bacillus there is created an environment favorable for the development of organisms of the Friedlander group, and these in turn exercise an action antagonistic to the Perez organism. There are many parallels for the assumption of this attribute of the Friedlander bacilli.

In a considerable series of studies of the nasal bacterial flora of nonozena cases, the Perez coccobacillus was never found, except in three cases of syphilitic ozena. These cases, though, were typical ozena. Organisms of the Friedlander group were not infrequently present.

Again, Abels bacilli vaccins are without the slightest influence in Abels ozena cases, but the focal response in these cases under Perez vaccin therapy is startling.

The theory that dogs are the medium of fetid ozena transmission has been both exploited and questioned. Up to the present time we have given no consideration to this phase of ozena, and have taken bacillus bronchisepticus for comparative study, as it is the organisms more like coccobacillus ozenæ fetidus than any other of which we have knowledge. Distemper, a common and often an epidemic canine malady, begins as a nasal infection. While kennels may at times be decimated, the animals usually recover. Infection produces prostration, but death is usually due to secondary invaders. Distemper carriers are not common.

Résumé.—Some characteristics of coccobacillus fetidus ozenæ (Perez) have been heretofore erroneously described, in that the organism is motile, may ferment carbohydrats and may produce acid.

Coccobacillus ozenæ fetidus and Abel's bacillus are distinct bacterial entities.

There is a startling similarity between coccobacillus ozenæ fetidus and bacillus bronchisepticus, the specific organism of canine distemper. This similarity is pronounced in complement fixing phenomenon.

The coccobacillus fetidus ozenæ group is made up of many subvarieties or strains.

Coccobacillus fetidus ozenæ is probably the specific organism of typical fetid ozena in man.

A change in the name of the organism from coccobacillus ozenæ fetidus to bacillus rhinosepticus is proposed.

GENERAL CONCLUSIONS.

The conclusions stated in our preliminary report, with exceptions about to be noted, still stand, and we repeat them here:

1. The coccobacillus fetidus ozenæ Perez, as isolated by Hofer, has answered all the bacteriologic requirements necessary to establish its identity as the etiologic factor in ozena.

(Note.—We feel that this conclusion must be accepted with certain reservations. We have been unable to obtain a complement fixation with untreated ozena patients. Whether this failure is due to faulty technic or, for various reasons, impossible of accomplishment, we are unable to state.)

2. The isolation of this organism is attended with considerable difficulty.

(Note.—This statement no longer holds, as with our discovery of the motility of the organism, our improved methods of isolation, and the ease with which the cultures can be proved by agglutination and complement fixation reactions, the matter is comparatively a simple one.)

3. The production of agglutinating serum in rabbits is an exceedingly difficult task.

(Note.—The use of live instead of killed cultures has enabled us with as few as four injections, and within a period of four weeks, to produce a serum which agglutinates in a dilution as high as one to two thousand.

4. The preparation of autogenous vaccins in every case is very difficult, if not impossible.

(Note.—This statement refers to the difficulty of isolating the Perez organism in the Friedlander group. Now that we have found that almost as brilliant results can be obtained in this group by means of our stock Perez vaccin, this difficulty in the treatment has been overcome.

5. At present mixed vaccins made from various strains of

Perez bacillus is the most practical method of treatment which is now available.

(Note.—This statement stands without change.)

6. It may be necessary to precede or combine with the treatment the vaccins made from the organisms which are usually present in combination with the Perez bacillus.

(Note.—As already noted, the preliminary treatment with a mixed stock vaccin has proved unnecessary. In our first vaccins we combined the Perez bacillus with the Friedlander, micrococcus catarrhalis, pneumococcus, and other well-known organisms found in chronic catarrhal conditions, and found very quickly that these mixed vaccins were not nearly as efficacious as vaccins made from the various strains of the Perez organism.

7. It may be possible that there may be two or more types of ozena, bacteriologically different but clinically identical.

8. In spite of the presence of organisms of the Friedlander group, in cases of undoubted clinical ozena, we consider that these organisms have no etiologic significance.

(Note.—As already noted, we have, for the sake of convenience, divided clinical ozena into two bacteriologic groups. With improved technic and further morphologic studies, we believe the socalled Friedlander group will become increasingly smaller.)

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CHART I.

Bacteriologic Findings in Cases of Rhinitis Atrophicans Nonfetida.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Examination	Odor	Crusts	Notes.
1	C. K.	18	M.	Jew	Office	Antrum and frontal sinus suppuration	Staphylococci	0	0	No improvement with autog. vaccin.
2	C. W.	20	M.	American	Poly.	Rhinitis Atrophicans	Staphylococci	0	0	
3	V. D.	35	F.	American	Poly.	Rhinitis Atrophicans	Staphylococci	0	0	Empyema antrum.
4	E. U.	25	F.	American	Poly.	Rhinitis Atrophicans	Staphylococci	0	+	
5	S. T.	27	F.	German	Poly.	Rhinitis Atrophicans	Staphylococci	0	0	
6	S. C.	26	F.	American	Nise	Rhinitis Atrophicans	Pneumococci	+	+	Case reported improved with autog. vaccin.
7	O. O.	?	?	American	Lewitt	Rhinitis Atrophicans	Micrococcus catarrhalis	+	+	Odor not typical.
8	McG.	31	F.	American	Poly.	Rhinitis Atrophicans	B. pyocyaneus	0	0	

9	G. M.	6	M.	American	Poly.	Rhinitis Atrophicans	Pneumococcus	O	O	
10	M. J.	30	M.	American	U. C.	Normal Mucous Membrane	Staphylococci	O	O	Father of case 30, Chart II. Congenital syphilis.
11	F. J.	5	F.	American	U. C.	Normal Mucous Membrane	Staphylococci	O	O	Daughter of M. J.
12	B. J.	6 mos.	F.	American	U. C.	Normal Mucous Membrane	Staphylococci	O	O	Daughter of M. J.
13	E. J.	30	F.	American	U. C.	Normal Mucous Membrane	Staphylococci	O	O	Wife of M. J.
14	J. G.	50	M.	French	Office	Rhinitis Atrophicans	Micrococatarrhalis	O	O	
15	H. G.	54	M.	American	Office	Rhinitis Atrophicans	Staphylococci	O	O	

CHART II.

Perez Group. All Cases True Clinical Ozena. General Analysis.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Finding	Agglutination	No. of Inject's	Crusts	Odor	Result.
1	Adolphus	28	M.	German	Victors	Ozena	Perez	+	20	+	+	No crusts, no odor. Clinical cure. Under treatment.
2	Alpert	22	M.	Russian Jew	Richards	Ozena	Perez	+	10	+	+	No crusts, no odor. Clinical cure.
3	Evadikin	25	F.	Russian Jew	Hutchins	Ozena	Perez	+	10	+	+	Not treated by me. Reported improvement marked.
4	Femol	16	M.	?	Hunt	Ozena	Perez	+	?	+	+	Not treated by me. No report to date.
5	Faber	33	M.	French	Poly.	Ozena	Perez	+	6	O	+	No crusts, no odor. Clinical cure, patient's standpoint.
6	Griffin	21	F.	American	Green	Ozena	Perez	+	15	+	+	No crusts, no odor. Clinical cure.
7	Hillman	21	F.	American	Office	Ozena	Perez	1	15	+	+	No crusts, no odor. Clinical cure.

8	Kelleher	17	F.	German	Victors	Ozena	Perez	+	20	+	+	No crusts, no odor. Clinical cure. Under treatment.
9	Luoto	40	M.	German	Stanford	Ozena	Perez	+	7	O	+	No crusts, no odor. Under treatment.
10	Miranda, Mrs.	37	F.	Spanish	Poly.	Ozena	Perez	+	9	+	+	No crusts, no odor. Under treatment.
11	Miranda, J.	11	M.	Spanish	Poly.	Ozena	Perez		9	+	+	No crusts, no odor. Under treatment.
12	Miranda, H.	12	M.	Spanish	Poly.	Ozena	Perez		9	+	+	No crusts, no odor. Under treatment.
13	Novani	14	F.	American	Office	Ozena	Perez		12	+	+	No crusts, slight odor. Stopped treatment.
14	Plant	20	F.	American	Office	Ozena	Perez	+	19	+	+	No crusts, no odor. Clinical cure.
15	Piffle	30	M.	American	State Insane Asylum	Ozena	Perez	+		+	+	No treatment.
16	Spencer	?	M.	?	Spencer	Ozena	Perez	+		+	+	No treatment.
17	Stefanovich	25	M.	Servian	Stanford	Ozena	Perez		10	+	+	No crusts, no odor. Clinical cure.

CHART II. (Continued)

Perez Group. All Cases True Clinical Ozena. General Analysis.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Finding	Agglutination	No. of Inject's	Crusts	Odor	Result.
18	S. L.	16	F.	American	Johnson	Ozena	Perez	+		+	+	Meagre reports, great improvement. Not seen by me.
19	Strum	19	F.	American	Hutchins	Ozena	Perez	+	10	+	+	Some crusts, some odor. Great! improvement. Not seen by me.
20	Spooner	35	M.	American	Office	At Rhin.	Perez	—	5	O	O	Ozera larynx.? Great improvement.
21	Pris	22	F.	German	Office	Pan Sinusitis	Perez	—	19	+	+	No crusts, no odor. Clinical cure.
22	Weber	27	M.	American	Office	Ozena	Perez	+	14	+	+	No crusts, no odor. Clinical cure.
23	Zamla	25	F.	Hungary	Office	Ozena	Perez		3	+	+	Marked improvement. Did not return.

24	De Vecci	18	F.	Italian	Poly.	Ozena	Perez			+	+	No treatment.
25	Spicer	?	?	?	Spicer	Ozena	Staph			+	+	No treatment.
26	Smith	22	F.	American	Benedict	Ozena	Staph			+	+	No treatment.
27	C. K.	13	M.	American	Maxey	Ozena	Staph			+	+	No treatment.
28	Jolley	30	M.	Russian Jew	Poly.	Ozena	Staph		7	+	+	No crust, no odor. Clinical cure, patients standpoint.
29	Morgan	21	F.	American	Stanford	Ozena	Perez		4	+	+	No crust, no odor. Clinical cure, patients standpoint.
30	Jackson	9	F.	American	U. C.	Ozena	Perez	+	O	+	+	Congenital syphilis. No treatment.
31	J. W.	50	M.	American	U. C.	Ozena	Perez	+		+	+	Active syphilis. No treatment.
32	E. H.	37	M.	American	U. C.	Ozena	Perez	—		+	+	Active syphilis. No treatment.

CHART III.

Clinical Ozena. Friedlander Group. Treated with Perez Vaccine. General Analysis.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Examination	Odor	Crust	No. of Injections	Agglutination with Perez Strains	
1	Walsh	23	F.	American	Poly.	Rhinitis atrophicans nonfetida	Friedlander	O	O			
2	Rosberg	18	F.	American	Poly.	Rhinitis atrophicans nonfetida	Friedlander	O	O			
3	Ticox	22	M.	American	Nagner	Ozena	Friedlander	?	+			
4	Tuckey	30	M.	American	Office	Rhin. Atroph.	Friedlander	O	O			Several examinations showed organism closely resembling Perez.
5	Ortereras	30	F.	Mexican	Poly.	Rhin. Atroph.	Friedlander	O	O		Neg.	
6	Kenealy	35	F.	American	Office	Rhin. Atroph.	Friedlander	O	+		Neg.	History of typical ozena in childhood. No odor for years. Pansinuitis right side.
7	Gross	26	M.	American	Jordan	Ozena	Friedlander	+	+	?	Neg.	Case reported greatly improved by Perez Vaccin.

8	Grove		F.	American	McLeod	Ozena	Friedlander	+	+	?	Neg.	Case reported greatly improved by Perez Vaccin.
9	Cortelyou	30	F.	American	Hopkins	Ozena	Friedlander	+	+		Neg.	
10	Belvol	7	F.	American	U. C.	Ozena	Friedlander	+	+		Neg.	Treated over year at U. C. with autog. vaccin. Unsatisfactory results.
11	Bunw	25	F.	English	Stanford	Ozena	Friedlander	+	+	16	Neg.	Clinical cure. No crusts, no odor.
12	Chun	14	F.	Chinese	Stanford	Ozena	Friedlander	+	+	13	Neg.	Clinical cure. No crusts, no odor.
13	Dougherty	14	M.	American	Stanford	Ozena	Friedlander	+	+	6	Neg.	Great improvement under treatment.
14	Devino	30	F.	Italian	Stanford	Ozena	Friedlander	+	+	13	Neg.	Great improvement under treatment.
15	Mahan	13	F.	American	Office	Ozena	Friedlander	+	+		Neg.	Great improvement under treatment.
16	Ortereras	8	M.	Mexican	Office	Ozena	Friedlander	+	+	11	Neg.	Great improvement under treatment.
17	Ortereras	7	M.	Mexican	Office	Ozena	Friedlander	+	+	7	Neg.	Great improvement under treatment.
18	Posatani	6	M.	Italian	Stanford	Ozena	Friedlander	+	+	10	Neg.	Great improvement under treatment.
19	Parenti	28	M.	Italian	Stanford	Ozena	Friedlander	+	+	9	Neg.	Great improvement under treatment.

CHART IV.

Analysis of Cases of Syphlitic Ozena.

Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Examination	Wassermann	Type of Syphilis	Odor	Crusts	1st. Ex.	Last Ex.	Result of Anti-Syp. Treat.
19954 J. B.	4	M.	American	U. C.	Syph. Ozena	Staph.	+++	Active Congenital	O	S	12-25-15	4-10-16	No change.
19161 J. W.	50	M.	American	U. C.	Syph. Ozena	Perez +	+++	Ter.	O	O	12-25-15	4-10-16	No change.
11055 A. M.	35	F.	Russian	U. C.	Syph. Ozena	Staph.	+++	Ter.	+	+		4-10-16	
11785 E. G.	37	M.	Italian	U. C.	Syph. Ozena.	Fried.	+++	Tabes.	+	+	12-25-15		
18821 A. J.	9j	F.	American	U. C.	Syph. Ozena	Perez +	+++	Active Congenital	+	+	12-25-15	4-10-16	No change.
19102 R. E.		F.	American	U. C.	Syph. Ozena	Staph.	+++		O	+	12-25-15		
19709 E. H.	37	M.	American	U. C.	Syph. Ozena	Perez +	——	Sec. Active	O	+	12-25-15	2-6-16	No change.
19348 A. G.	25	M.	Italian	U. C.	Syph. Ozena	Gram-Bac?	+++	Sec. Active	+	S	12-25-15		

CHART V.

Showing Results of Treatment with Perez Vaccine on Cases of Clinical Ozena, Friedlander and Perez Type.

No.	Name	Bac. Ex.	1st. Inj.	Last Inj.	No. of Inj.	Notes, 1st Ex.	Notes, Last Inj.	Patient's statement as to improvement in general condition	Notes
1	Miranda J.	Perez.	Apr. 10, 1915	Apr. 12, 1916	12	Sept. 22, 1914 Greenish crusts, odor offensive	April 12,1916 No crusts, no odor	General condition much improved	Stayed away one year, odor better as whole but nose must be washed from time to time. Clinically improved, but not cured. Still under treatment.
2	Miranda H.	Perez.	Apr. 13, 1914	Apr. 12, 1916	9	Apr. 13, 1914 L. nostril completedly blocked, odor very marked	April 12, 1916 No crusts, no odor	General condition much improved	Notes as with M. J.
3	Novani	Perez.	July 26, 1915	Oct. 9, 1915	12	July 26, 1915 Typ. ozena, crusts in entire nose	Oct. 9, 1915 No crusts, slight odor	General condition much improved	Pat. father does not believe in vaccines, admits great improvement.

CHART V. (Continued)

No.	Name	Bac. Ex.	First Inj.	Last Inj.	No. of Inj.	Notes 1st Exam.	Notes Last Exam.	Patient's statement as to improvement in general condition	Notes
4	Plant	Perez.	July 8, 1915	Feb. 4, 1916	26	July 8, 1915 Heavy crusts, foul sweetish odor.	Feb. 4, 1916 No crusts, no odor	Mental condition much improved. General condition vastly changed for better.	Discharged Feb. 4, 1916, clinical cure. Injections stopped for present, no odor since Jan. 11, 1916.
5	Stefanovich	Perez.	March 9, 1916	Apr. 12, 1916	11	March 9, 1916 Slight crusts, odor strong at 4 ft.	April 12, 1916 No crusts, no odor	General condition much improved.	Clinical cure, discharged.
6	Spooner	Perez.	Feb. 18, 1916	Feb. 26, 1916	5	Feb. 18, 1916 No clinical ozena in nose. Ozena of larynx, slight crusts and redness.	Feb. 26, no crusts in interarytenoid space, muc. mem. distinctly reddened and not so shiny.	Subjective improvement in larynx marked.	Discharged for 3 mos.
7	Sister P.	Perez later Fried.	Apr. 5, 1915	Nov. 15, 1915	20	Aug. 6, 1911 Pansinuitis, crusts odor, various operations.	Nov. 10, 1915 In treatment 4 years, no pus, crusts or odor.	Tremendous improvement.	Clinical and bacteriologic cure probable, repeated bac. ex. Perez neg.
8	Weber	Perez	Dec. 24, 1915	Mar. 22, 1915	13	Dec. 24, 1915 Crusts slight, strong sweetish odor.	March 22, 1915 Perez neg. since March 3, no crusts, no odor.	Tremendous improvement.	Clinical and bacteriologic cure probable.

9	Jooley	Staph.	Feb. 16, 1916	Feb. 25, 1916	7	Feb. 16, 1916 True clin. ozena, crusts and odor	Feb. 25, 1916 No crusts, no odor	Tremendous improvement.	Did not return. Probably Perez infection. Clinical cure patient's standpoint.
10	Morgan	Perez	Mar. 9, 1916	Mar. 13, 1916	4	Mar. 9, 1916 Foul char. odor. Large crusts.	Mar. 13, 1916 No odor, few crusts.		Clinical cure patient's standpoint, local improvement wonderful.
11	Posatani	Fried.	Mar. 24, 1916	Apr. 12, 1916	7	Nose filled with black crusts, odor foul at 6 ft.	April 8, 1916 Slight odor and crusts. Contact.		Improvement remarkable. Under treatment.
12	Parenti	Fried.	Feb. 19, 1916	Mar. 14, 1916	9	No crusts, slight odor.	No crusts, no odor.	Much improved.	Did not return.
13	Devino	Fried.	Feb. 24, 1916	Apr. 13, 1916	10	Crusts l. only, odor marked, not typ.	No crusts, slight odor.		Under treatment. Results unsatisfactory.
14	Dougherty	Fried.	Mar. 28, 1916	Apr. 13, 1916	3	Nose blocked, extremely offensive.	No improvement.		Under treat. Results unsatisfactory.

CHART V. (Continued)

No.	Name	Bac. Ex.	1st Inj.	Last Inj.	No. of Inj.	Notes on 1st Ex.	Notes on last Ex.	Patient's statement as to improvement in general condition	Notes
15	Adolphus	Perez. Later Fried.	Dec. 15, 1915			Dec. 1, 1915 Typ. heavy crusts, foul, overpowering sweetish odor	April 2, 1916 No crusts, no odor	Very great increase weight and appetite, mentally better	Case still under treatment, clinical not bacteriological cure.
16	Alpert	Perez	Nov. 29, 1915	Feb. 8, 1916	9	Nov. 29, 1915 No crusts, foul sweetish odor.	April 3, 1916 Nose not washed for one month, no crusts, no odor.	Same statement as above.	To date, clinical but not bacteriological cure.
17	Faber	Perez	Mar. 11, 1916	Mar. 20, 1916	6	After 11 days without wash, few crusts, medium but characteristic odor.	March 20, 1916 No crusts, no odor.	Same statement as above.	Very irregular in attendance, permanent cure doubtful, clinical cure from patient's standpoint.
18	Griffin	Perez	June, 1915	Dec. 27, 1915	25	Violent frontal headache, crusts must be removed with instruments twice a week, odor unbearable to friends. Aug. 26, 1915	No odor, no crusts, Dec. 27, 1915	Same statement as above.	Clinical but not bacteriological cure. Case refuses further treatment.

19	Hillman	Perez	Aug. 13, 1915	Jan. 27, 1916	18	Aug. 13, 1915 Crusts slight, odor not noted, patient says odor is always complained of by surrounding.	Feb. 4, 1916 No odor, no crusts.	Same statement as above.	Apparently clinically cured.
20	Kelleher	Perez				July, 1915 Crusts completely block nose, foul overpowering odor.	April 2, 1916 No crusts, no odor.	Same statement as above.	Clinical cure. Case still under treatment.
21	Luoto	Perez	Apr. 8, 1916	Apr. 12, 1916	3	April 8. No crusts, no odor.	April 12, 1916 No crusts, no odor.		Clinical cure. Case still under treatment.
22	Miranda C.	Perez	Mar. 31, 1916	Apr. 12, 1916	4	March 31, 1916 Slight crust, no odor, formerly strong.	April 12, 1916 No crusts, no odor.		Clinical cure. Case still under treatment.

CHART VI.

General Survey of Rabbit Experiments to Obtain Agglutinating Sera.
Dosage: 5 cubic centimeters of salt solution added to 24 hour growth on agar and emulsion made.

No.	First Injection	Last Injection	Total No. of Injec.	Highest Dose	Live or Dead Cultures	Date of Death	Autopsy Condition of Turbinates only	Culture	Agglutination	Culture	Notes
1	Oct 17/14.	Oct. 23/14	2	0.75 c. c.	Killed	Oct. 30/14	Accidental death Ant. turb. distinctly congested.		Negative	Original Vienna	
2	Oct. 17/14	March 15/15	11	5.0 c. c.	Killed	March 16/15	Intense engorgement ant. end inf. turb.		Negative	Original Vienna	Lived six months. Death followed overdose.
3	Oct. 23/14	Nov. 4/15	3	2.0 c. c.	Killed	Nov. 23/15	Congestion ant. turb. Pus.	Perez neg.	Negative	Original Vienna	Killed by chloroform.
4	Oct. 23/14	Dec. 23/14	5	5.0 c. c.	Live	March 10/15		Perez neg.	Negative	Original Vienna	Killed.
5	Nov. 20/14	Dec. 23/14	4	2.0	Live	Feb. 23/15	Marked atrophy of turbinates.	Perez neg.	Negative	Original Vienna.	Average loss of weight 200 gm. between injections.
6	Dec. 7/14	Dec. 23/14	3	2.0	Killed	Jan. 2/15	Congestion of turb.	Perez neg.	Negative	Original Vienna	Death due to general Perez infection.

7	Dec. 15/14	Dec. 23/14	2	2.0	Killed	Dec. 23/14	Intense congestion	Perez neg.		Original Vienna	Death from fatal dose, 12 hours.
8	Dec. 15/14	Dec. 15/14	1	2.0	Killed	Dec. 15/14	Intense congestion.	Perez +?		Original Vienna	Death from fatal dose, 12 hours.
9	Dec. 16/14	April 1/15	2	2.0	Killed	April 1/15				Original Vienna	Death from fatal dose, 14 hours.
10	Nov. 27/14	Nov. 27/14	1	1.5	Killed	Nov. 27/14	Intense congestion			Original Vienna	Death from fatal dose, 8 hours.
11	Dec. 16/14	March 15/15	3	0.75	Killed	March 15	Intense congestion.			Vienna	Death from fatal dose, 12 hours.
12	Dec. 22/15	Dec. 27/15	1	0.5	Live	Dec. 27/15				Griffin	Death from fatal dose, 4 hours.
13	Dec. 22/15	Feb. 21/16	8	1.0	Live	Mar. 11/16	Bluish black marked atrophy nose filled pus.	Perez +	Strongly positive	Original Vienna	Lived two and a half months. Death from general Perez infection.
14	Dec. 22/15	March 15/16	13	1.0	Live	Feb. 19/16			Strongly positive	Original Vienna	Lived two and a half months. Death from general Perez infection
15	Dec. 22/15		1	0.2	Live	Dec. 22/15				Plant	Fatal dose. Death in 4 hours. Baby rabbit, weight 230 grams.
16	Dec. 22/15		1	0.2	Live	Dec. 25/15				Strum	Fatal dose. Death in 4 hours. Baby rabbit, weight 250 grams.

CHART VI.

General Survey of Rabb t Experiments to Obtain Agglutinating Sera.
Dosage: 5 cubic centimeters of salt solution added to 24 hour growth on agar and emulsion made

No	First Injection	Last Injection	Total No. of Injec.	Highest Dose	Live or Dead Cultures	Date of Death	Autopsy Condition of Turbinates only	Culture	Agglutination	Culture	Notes
17	Dec. 22/15	Jan. 21/16	7	2.3	Live	Feb. 4/16	Marked atrophy	Pure Perez	Strongly positive	Adolphus	Cause of death unknown. Apparently in good condition.
18	Jan. 21/16		1	0.4	Live	Jan. 23/16	No change.			Alpert	Cause of death unknown.
19	Dec. 27/15		1	0.2	Live	Dec. 27/16	Acute conges.			Femol	Fatal dose. Died 4 hours. Baby 230 grams.
20	Jan. 7/16	Jan. 10/16	2	0.6	Live	Jan. 13/16	Slight conges.	Staph.		Griffin	Fatal dose. Baby, 2½ months.
21	Jan. 7/16	Jan. 27/16	4	0.8	Live	Feb. 12/16		P. C. Perez	Negative	plant	Baby, 2½ months. Lived 3 months.
22	June 15/15		1	5.0	Live	Dec. 6/15		P. C. Perez	Positive	Vienna	Survived one enormous dose of 24 hour agar growth.
23	Jan. 7/16	Jan. 10/16	2	0.6	Live	Jan. 17/16	Destruction of turb.	P. C. Perez	Positive	Femol	Baby, 2½ months. Greatly emaciated at death.

24	Dec. 22/15	Dec. 31/15	8	1.0	Live	Feb. 7/16			Positive	Kelleher	Doses given at four day intervals.
25	Dec. 27/15		1	0.2	Live	Dec. 30/15		P. C. Perez	Positive	Vienna	Baby, 230 grams. Acute coryza following inj.
26	Jan. 10/16		Inoculation of nose		Live			P. C. Perez	Positive	Vienna	Baby, 2½ months. Exp. for direct transference. Still living.
27	Jan. 10/16		Inoculation of nose		Live			Perez negative		Vienna	Baby, 2½ months. Exp. for direct transference. Still living.
28	Dec. 10/15		1	0.2	Live	Dec. 11/15				Vienna	Baby, 244 grams. Ex. to det. size of fatal dose.
29	Dec. 31/15		1	0.5	Live	Dec. 31/15	Intense congestion.			Plant	Died in 4 hours; fatal dose.
30	Dec. 31/15		1	0.4	Live	Dec. 31/15				Jackson	Died in 4 hours; fatal dose.
31	Dec. 31/15		1	0.5	Live	Dec. 31/15	Intense congestion.			Griffin	Died in 4 hours; fatal dose.
32	Dec. 10/15		1	0.5	Live	Dec. 10/15				Vienna	Died in 7 hours; fatal dose.
33	Dec. 31/15	Feb. 9/16	8	1.0	Live	Feb. 21/16			Positive	Femol	Baby, 1st dose 0.1 c.c. increased by 0.1 each dose. Last dose fatal.

CHART VI.

General Survey of Rabbit Experiments to Obtain Agglutinating Sera.
Dosage: 5 cubic centimeters of salt solution added to 24 hour growth on agar and emulsion made

No.	First Injection	Last Injection	Total No. of Injec.	Highest Dose	Live or Dead Cultures	Date of Death	Autopsy Condition of Turbinates only	Culture	Agglutination	Culture	Notes
34	Dec. 7/15	Dec. 7/16	3	0.6	Live	Dec. 7/16		P. C. Perez	Positive	Mixed	Strong agg. serum after 2nd inj. Wt. 1340 grams.
35	Dec. 3/15	Jan. 21/16	3	0.3	Live	Jan. 21/16	Slight congestion	P. C. Perez	Positive	Vienna	Baby rabbit.
36	Nov. 29/15	Dec. 23/15	3	2.0	Killed	Dec. 20/15	Slight congestion	P. C. Perez	Positive	Alpert	Lost 200 grams after two inj.
37	Nov. 29/15	Dec. 13/15	3	1.0	Killed	Dec. 13/15				Griffin	Fatal dose.
38	Dec. 13/15	Dec. 17/15	2	0.4	Live	Dec. 25/15	Atrophy and pus pockets	P. C. Perez	Positive	Vienna	Strong agglutination, even with two injections. Reduced to skeleton.
39	Jan. 27/16	Mar. 29/16	8	1.4	Live	April 14/16	Atrophy and pus.	P. C. Perez	Positive	Vienna	Killed.
40	Jan. 27/16	Feb. 14/16	5	2.0	Live	Feb. 25/16	Atrophy and pus.		Positive	Vienna	Killed.
41	Jan. 27/16	Feb. 14/16	5	2.0	Live	Feb. 25/16			Positive	Alpert	Killed and bled. Strong agglutination.

42	Jan. 27/16	Feb. 14/16	5	1.8	Fried-lander.				Negative	Eugene O.	No agglutination for any Fried. strains. Rabbit living and well.
43	Jan. 31/16	Mar. 15/16	7	2.0	Perez, Live				Positive	Jackson	Agglutination strong after fourth injection.
44	Feb. 9/16	Feb. 25/16	4	1.5	Perez, Live					Piffle	Killed and bled. Agglutination weak.
45	Feb. 9/16	Mar. 29/16	7	2.0	Perez, Live	April 24/16				Kelleher	
46	Feb. 14/16	Mar. 15/16	6	2.0	Perez, Live					Jackson	Rabbit living.
47	Feb. 21/16	Mar. 3/16	4	2.0	Perez, Live	Mar. 12/16				Plant	Died; no autopsy.
48	Feb. 21/16	Mar. 29/16	6	2.0	Perez, Live					Adolphus	Rabbit living.
49	Feb. 21/16		1	0.5	Mixed Live	Feb. 22/16				Kenealy	Friedlander and Streptococcus.

XI.

EXPERIENCE WITH SUSPENSION LARYNGOSCOPY IN OVER TWO HUNDRED CASES, WITH REPORT.*

By Joseph C. Beck, M. D.,

Chicago.

To criticise fairly any new method, one should have sufficient experience in order neither to be overenthusiastic and so mislead men into abandoning the well-tried and successful methods, nor to report discouragingly and so engender at the outset prejudice against giving a new procedure a trial. Having previously made this same mistake, and being aware of others who have done likewise, I have purposely avoided the publication of our results with suspension laryngoscopy until this day, after three years' experience in more than two hundred cases in private practice.

We are again indebted to Killian for a great contribution to science and to laryngology in particular; and I wish to say, at the outset, that I believe suspension laryngoscopy is destined to become one of the greatest helps in the diagnosis and surgical management of diseases of the larynx, trachea, bronchi and esophagus.

The evolution of the method from its first accidental discovery while Killian was working on a cadaver, up to the present time, when Lynch has perfected the instrumentarium, is most interesting and can be found in the new edition of Jackson's textbook on Peroral Endoscopy and Laryngeal Surgery.

It is to Freudenthal that I acknowledge my first clear information on this subject, when he demonstrated the method before the American Laryngological, Rhinological and Otological Society, three years ago. With the first type of Killian's instruments (Figure 1) I began experimenting in the exam-

*Read before the Chicago Otolaryngological Society, November 16, 1915.

ination of various cases, and found it most difficult and unsatisfactory. I was only able to succeed with general anesthesia, and even then not with absolute satisfaction. One afternoon, while demonstrating the method to Dr. Lynch, who was visiting me, I noted his criticism and enthusiasm, and in less than six months I found, to my great satisfaction, that most of my first objections were overcome by his perfection of instruments and technic, as well as by his addition of many new features, which he demonstrated by treating a number of patients with particularly good results.

During the progress of Lynch's work, a number of European articles appeared as well as newer instruments, particularly those of Albrecht, and Bruning's counter pressure attachment (Figure 2), which I found to be an improvement on Killian's original (Figure 1), as well as on Killian's improved instrument (Figure 3).

Until I obtained the last model of Lynch's instrument (Figure 4), the greatest difficulties with us have always been: (1) To prevent the tongue from sliding to one side or the other; (2) to keep the epiglottis on the point of the spatula; and (3) our inability to expose the anterior commissure in every case. I also noted that following the use of the apparatus the patient complained considerably of soreness, or at least of an uncomfortable feeling at the base of the tongue and in the teeth.

Several patients I was unable to suspend on account of certain pathologicoanatomic conditions, such as: (1) The presence of long upper central incisors, there being no other teeth present in the upper jaw; (2) a stiff neck from an old cured scoliosis, preventing extension of the neck; (3) a tumor at the base of the tongue in the region of the valecula; (4) complete absence of the teeth in the upper jaw, with a great deal of absorption of the alveolar process; (5) very loose teeth in patients with pyorrhea alveolaris; and (6) partial ankylosis of the lower jaw. In addition to these there were some individuals who, in spite of the most thorough local anesthesia, would continue to react and thus foil any attempt at suspension. These were probably due to mental anxiety.

Some of the conditions in which we have employed suspension laryngoscopy are: (1) Carcinoma, (2) adenocarcinoma, (3) papilloma, (4) fibroma, (5) angioma, (6) syphilis,

(7) tuberculosis, (8) rhinoscleroma, (9) cicatricial stenosis, (10) paralysis, (11)) foreign bodies, (12) burns, (13) asthma, (14) posttraumatic tracheal stenosis, (15) bronchiectasis, (16) suction of secretion for bacteriologic examination, (17) direct intubation, (18) simple laryngitis, (19) posttyphoidal chondritis, (20) atrophic laryngotracheitis, (21) hysteric aphonia, (22) suspected thymus enlargement, (23) tracheal compression by thyroid gland or tumor, (24) aortic aneurism, (25) normal cases.

INSTRUMENTS AND TECHNIC.

Instruments.—(1) Table with the Lynch extension (Figure 5), (2) crane, (3) hook, (4) spatulæ, (5) illumination (Beck's lamp) (Figure 6).

Technic.—(1) Anesthesia. This may be local by applying cocain twenty per cent and injecting novocain one per cent into superior laryngeal nerve; or general by the use of ether vapor.

(2) Position of patient. Patient is in recumbent position, with head in extension over edge of table.

(3) Introduction of instrument as far as epiglottis, then under it—i. e., on its ventricular surface.

(4) Counter fixation on the upper incisors by the tooth plates.

(5) Opening of mouth to a space sufficient for working purposes.

(6) Hook is placed on the transverse bar of the crane, while patient's head is kept well in the median line.

(7) Horizontal bar is raised by turning the vertical portion of the crane, thus lifting head off the table.

(8) The larynx and hyoid bone are brought forward and further upward by turning the handle of the horizontal portion so that the latter moves toward the operator.

(9) The larynx is fixed and the anterior commissure brought more into view by pressing the thyroid cartilage down with the left hand, in preference to the Bruning's counter pressure instrument.

(10) The right hand is left free for any manipulations in the matter of treatment, diagnosis, operations or further manipulation of the crane. For the removal of tumor, either for diagnostic or operative purposes, we have found that the

Cordes instrument with a set of five tips (Figure 7) has served us the best. One should always have on hand a substitute, and one of the best is the Killian short alligator bronchoscopic forceps (Figure 8). A long handled ring curette (Figure 9) and a long dressing forceps for sponging or removing particles of tissue (Figure 10) make up the instrumentarium. The elaborate and well devised set of instruments of Dr. Lynch are unquestionably of value, especially in the hands of such an expert as himself; but we have been able to manage with those mentioned.

We have never sutured any part of the larynx from within under suspension, but we have made use of the actual cautery in several cases of ulceration, especially of the tuberculous type, and we found a long electrode with especially heavy blade of greatest service (Figure 11). The suction apparatus is of great assistance in the work, and an atomizer with cocain and adrenalin should always be on hand, in order to supplement the anesthetic while working with the local anesthesia. Morphin and atropin are excellent accessories to good work.

When general anesthesia is required, it is well to employ the vapor method; and ether is the drug of choice, although we have used nitrous oxid and oxygen in one case, and ether and oil per rectum, also scopolamin—morphin in several other cases. In a number of cases in which tracheotomy had been performed previously or at the time of the suspension procedure, the intratracheal method of administering ether or chloroform was employed.

CASE REPORTS.

Case 1.—Mr. M., forty-six years of age, was referred to me by a laryngologist, with the diagnosis of adenocarcinoma. The doctor had removed a piece of tissue for microscopic examination, and this, with the pathologist's report, was furnished by the patient. Symptoms were hoarseness and some embarrassment in breathing. General condition was good, although the patient had lost some weight.

Examination under suspension laryngoscopy revealed a small growth, irregular in outline, situated on the left vocal cord, near the anterior commissure. Arytenoids moved freely, and there was no enlargement of cervical glands. A piece of tissue was removed, and all the physical and laboratory tests,

including the Abderhalden, were made. Wassermann was negative, Abderhalden positive; physical examination was negative. Microscopic diagnosis of the pieces of tissue removed was adenocarcinoma.

Treatment.—Twelve deep X-ray treatments, by means of the Coolidge tube, were given over different areas of the larynx within two weeks. Then by means of suspension laryngoscopy and the Cordes biting forceps (Figure 7), all the growth was removed. Two days later the deep X-ray treatments were again resumed. About one week later the patient exhibited symptoms suggestive of hyperthyroidism, and he was given thyroid extract tablets, which he has been taking for four months according to indications.

After these procedures, reexamination of the larynx showed a complete disappearance of the adenocarcinomatous growth. Patient's voice was clear, and breathing was normal. At this time he went East for a two months' vacation, so I advised him to consult Dr. Edgar Holmes, from whom I received the following report:

"It was a pleasure to see your laryngeal malignant patient, especially as the present results seem so brilliant. I sincerely hope, for his sake, that there will be no recurrence. His phonation seems to cause so slight strain and little clanging of cords, that his chances seem much better than usual."

When the patient returned to see me again I found that there was no recurrence, and after two more weeks under observation he left in excellent condition.

The following telegram was just received from the patient:

"Nanaimo, November 13, 1915.

"Boucher away at war. Dr. Wilkes, who was treating me with Boucher, says no sign of growth. Good movement; practically no swelling or congestion; excellent voice; throat could hardly be better. Feel very well; weight about same as when in Chicago. Writing tonight. C. R. MASTER."

Case 2.—Master Kty., a boy, seven years old, who had been hoarse since infancy and recently complained of difficulty in breathing.

Examination by means of the laryngeal mirror, which was very easily performed, revealed a typical papillomatous mass filling the subglottic space, including the cords. An operation was decided upon and performed. Suspension was employed,

but as it was impossible to perform it under local anesthesia a general anesthetic was administered. As considerable difficulty in breathing resulted, it was decided to do a tracheotomy, which would also facilitate the healing and operation. While doing the tracheotomy the boy vomited a large quantity of thick material consisting of potatoes, etc., and we were fortunate in having at hand the suction apparatus to prevent inspiration of the vomitus. As soon as the tracheotomy was made, he was given the vapor ether anesthesia by that route. He was then very readily suspended and the growth removed. It was shown subsequently to be a papilloma by microscopic examination. Patient was put to bed in good condition. He had a fairly good night, but in the afternoon of the next day it was noted that his respiration increased to forty and his temperature to 101.6° with some cough. He complained of some pain in the left precordial and upper left abdominal region. It was also noted that he had practically no pulse on the left side, whereas on the right radial side his pulse was very distinctly felt. A probable diagnosis of bronchopneumonia was made by Dr. Meyers, and patient was placed under suitable treatment for such condition. The next day, the third after the operation, I saw him and found practically the same condition as described above, and in consulting with Dr. Friedberg decided that he probably had a pneumonia, due, I thought, to inspiration of some food particles, but Dr. Friedberg attributed it to the anesthetic. That evening when I left the hospital he had rallied some. A special nurse was put on the case, and she telephoned me about nine o'clock that his temperature had gone down to a little over one hundred, and he appeared to breathe easier. About ten-thirty o'clock he asked her for some drinking water, and the next moment she noticed a sudden gush of blood from the tube and the mouth. The quantity of blood was considerable. He immediately sank and died in a few moments, in spite of heroic efforts to save the child. Postmortem examination was denied. It is a question why he had a pulse only on the right side and not on the left. It is not likely that it was an anomalous course of the radial, because he had been in the hospital for a few days before, and this no doubt would have been discovered; besides, I inquired of the family physician who had taken care of the boy, and he never discovered this.

Case 3.—Mrs. M., who has been hoarse for nearly two years and under the care of Dr. Denman of Toledo, a laryngologist, who had been treating her for a growth on the vocal cord. This growth has been there for nearly two years. It was operated upon by direct laryngoscopy by Dr. Meyers of Ann Arbor, and a small piece removed, which was proven by the microscope to be a granuloma. It appeared to return very soon, and the patient then consulted Chevalier Jackson, who declared it to be most probably a fibroma, and had the patient desist from loud speaking for several months. While this treatment did not cause the growth to disappear, nor the hoarseness to improve, Dr. Denman did note that the growth, which was always of a cherry red color, became paler when patient employed whispering voice.

When I saw the case last July in Toledo I found a small, bright red colored neoplasm on the left vocal cord, near the anterior portion. It was decided to remove this growth, but the patient would not consent to having it done under local anesthesia, since she seemed to have had considerable pain during the previous manipulations. Dr. Denman, who employs nitrous oxid and oxygen anesthesia for most of his work, and has an expert to give it, urged me to try to remove this growth under suspension and gas anesthesia. I consented, and to our great surprise it was not at all difficult. The patient awoke immediately after the growth was removed and larynx inspected to see that all was well. She did not, however, utter a sound. I was sure that this was probably only transitory, because we had the same thing occur before. After about two weeks I received a letter from the doctor, stating that aphonia was no better, although the cords were smooth and their movements normal. Two months later the doctor brought the patient up because the voice had not returned, and since the voice was of a potential value to the patient, considerable anxiety was felt by all. I found that the cords looked good and their movements were normal, but the patient could not speak above a whisper. Still expressing my belief to the patient that she would speak, and advising the doctor to continue expectant treatment, the patient went home somewhat more satisfied. Yesterday I received the following telegram, in answer to my inquiry:

"Toledo, Ohio, November 10, 1915.

"Dr. Joseph C. Beck: Cord smooth, straight, slightly congested; phonation most words possible, but difficult and fatiguing; quite hoarse; steady slow improvement.

"IRA O. DENMAN."

I have found no reference to this complication, but I believe that it is functional (hysteria) and not organic, since it occurred so rarely. It will, however, bear remembering and possibly study from another standpoint—namely, traction trauma, with possible muscular relaxation. The microscopic examination showed angiofibroma.

Case 4.—Mr. W., forty-two years of age. A very large healthy looking man. Hoarse for about six months. A laryngologist of his city, who referred him to me, stated that he observed the condition change frequently, whether under treatment or not. Examination showed the right arytenoid fixed and an irregular appearing growth on that side and somewhat subglottic, also involving part of the cord. By means of suspension a particle of the growth was removed, and the microscope showed it to be sarcomatous; yet the Abderhalden test for carcinoma was positive, while the Wassermann was negative.

Following the removal of the growth there resulted considerably more bleeding than normal, which continued for some time; so while the patient was suspended we introduced a large intubation tube, which controlled the hemorrhage beautifully.

EXHIBITION OF LARYNGEAL CASES, WITH SPECIAL REFERENCE TO SUSPENSION LARYNGOSCOPY.

The first case presented is a patient whose condition was diagnosticated as scleroderma by men who are supposed to be authorities in dermatology. Scleroderma is a condition in which there is this deformity in both hands, and probably also of the eye (Figure 12). Prof. Shamberg of Philadelphia has also found evidences in the larynx. In the case presented, the patient had a marked laryngeal obstruction that required tracheotomy, and subsequently laryngostomy was necessary. The cartilage was unusually hard, and there was difficulty in cutting through it. We were required to use bone forceps for this purpose. There is, as will be noticed when the man swallows, complete separation of the thyroid cartilage now. There

is an up and down tube placed into this slit (Figure 13), through which he breathes, one part going into the mouth, the other into the trachea. It is interesting that the condition of scleroderma occurs on the mucous membrane of the larynx, producing an irreparable condition and requiring such procedures as that performed in this case. Examination of the larynx from within simply shows the smooth surface of the vocal cords, with immovable arytenoids. There is still a plastic operation to be performed in this case after dilatation has been accomplished.

In splitting the larynx, after some few days, we noticed a white area, which we thought was caused by the pressure of the tube. For this reason we left the tube out for a few days, but found that this white, thick area on each side of the cricoid was still present. We excised it, and microscopic examination showed a hyperplasia of the mucous membrane and scar tissue. The case is too recent for any definite conclusions regarding it.

The second case is that of a man who came to the Cook County Hospital several years ago, requiring an emergency tracheotomy. He was fifty-six years of age, and had been hoarse for over three years. Although carcinoma was suspected in the case, he gave a very marked four plus Wassermann. He was given antisyphilitics, and subsequently a laryngostomy was performed. In closing the wound the plastic method was employed, using the skin. Two lateral incisions were made, the skin turned in and sutured in the median line. This patient has a defect in the thyroid cartilage, which can easily be felt. He is a laborer, and gets along very nicely, although there is still considerable difficulty in breathing when he has to exert himself. This case which is cured, is presented because of an interesting finding recently. In textbooks on dermatology and plastic surgery, it is said that whenever you transplant an area with hair into an area or region where there is mucous membrane, or where hair does not exist, the hair follicles die in the transplant. Nine months after the plastic in this case the man returned, complaining of a tickling in his throat. In the writer's absence, Dr. Pollock looked in his throat and found what he took to be a black thread there. The writer then suspended the patient and found that this black thread was a bunch of black hair, which could be pulled out easily. Microscopic examination substan-

tiates the presence of hair. To devise a method to destroy these hair follicles, if this hair returns, will be the next thing, and the X-ray is probably the only thing to be thought of at the present time. The man is gray haired, and yet the new hair is black. (Figures 14 and 15.)

The next patient is presented principally because it was impossible to suspend her, on account of the existence of a tumor at the base of the tongue. A woman, about thirty-five years of age, has had for several years the sensation as though there was something on the back of the tongue. In the past few months she had some difficulty in breathing and swallowing, as well as a change in her voice. These symptoms all increased in severity, so that the family physician referred her to a throat specialist, who in turn consulted me. Examination revealed a rounded looking tumor, smooth in character, about the size and shape of a half of a walnut and somewhat elastic but not painful on phonation. It did not involve the epiglottis, but pushed it backward so that it interfered with inspiration with a sort of valve action. I made a tentative diagnosis of thyroglossal duct cyst, and recommended puncture for diagnosis. We withdrew a very small quantity of a rose colored fluid, which the microscope showed contained a few red blood corpuscles; cultures remained sterile. We then decided that it was probably an accessory or internal thyroid gland, of which I have had one case, and decided upon operation. We believed that we could do so by the suspension method. On attempting to do so, however, we found it impossible, since the spatula could not be placed far enough back. I decided at that time to devise a spatula that would have a space in the center of its terminal or epiglottic end to permit the tumor to fit in and the side prongs of the spatula depressing the base of the tongue—a sort of a two-pronged fork—and thus enable one to employ suspension in similar cases. We did not wait for the construction of this spatula, however, but operated in the following manner, with excellent final results:

Patient was too irritable to do the operation under local anesthesia, so ether was employed. As soon as patient was anesthetized, it was found impossible to keep the tongue and, consequently, the tumor from closing off the larynx, so it became necessary to do a hurried tracheotomy through a very short fat neck. Subsequent anesthesia was administered

through this tracheal canula by the vapor method. It was soon discovered that the removal of the growth would be very difficult, if at all possible, by the oral route, so we determined to do an external pharyngotomy, which was comparatively easy of execution. The suction apparatus was particularly valuable for the removal of the blood and secretions of the pharynx. The tumor, which was very easily liberated from the tongue tissue proper, appeared to have a capsule. Its actual size is seen in Figure 16, and the macroscopic examination on cross section suggested a colloidal degenerated thyroid gland. Subsequent microscopic examination proved it to be such. The bleeding was easily controlled by a hypopharyngeal packing for twenty-four hours. The patient made an uneventful recovery, having some difficulty in swallowing, at first. She was kept on rectal feeding for five days, the tracheal tube was removed at the end of a week, and now one can see a smooth scar at the site of the previously located tumor. All the normal functions are preserved.

The next case is presented for many reasons of interest to the laryngologist. He was shown to you earlier in the evening when I showed the automatic closing of the tracheotomy tube. This man, who is about thirty years old, a machinist by trade, was first seen by me two years ago, when I found him suffering with chronic laryngeal obstruction of some sort, demanding urgent action to give him air. An emergency tracheotomy was performed and a correct diagnosis sought. At that period I left my service, and the patient came under the service of Dr. Stanton Friedberg. On my return, three months later, I found the patient in excellent general health. Dr. Friedberg informed me that he found some pus about the arytenoid region, and believed it to be some form of chondritis. He also stated that he made several punctures and got some pus. The most thorough and painstaking examination, as well as systematic treatment, excluded syphilis, tuberculosis, and laryngeal scleroma. We therefore decided on a laryngostomy, which was performed a little over one year ago. Under general vapor ether anesthesia, the thyroid cartilage was split. We found that the arytenoids were immobile, the vocal cords transformed into marked hypertrophic masses. This same type of thickened mucous membrane extended down through the larynx and trachea, almost within an inch of the sternal

notch, and so narrowed these passages as practically not to permit the introduction of a bougie one-eighth of an inch in diameter. The trachea, therefore, was also split down to the limits of this constriction. An up and down laryngostomy tube was inserted, and the larynx and trachea incisions kept apart by packing. Daily changing of the tube and redressing of the cleft for about three weeks showed that this above mentioned hypertrophic mucous membrane did not seem to disappear—on the contrary, it became thicker. We then decided to remove part of it, anticipating healing by a cicatricially modified epithelial lining. Under local infiltration anesthesia a portion was removed with considerable difficulty, on account of the extreme hypersensitiveness of the area; in fact, this was evident at each dressing, in spite of the very free use of a strong cocain solution. The up and down tube was again introduced and the laryngotracheal cleft kept apart for several weeks. During this time it developed, from the examination of his nose, that he had a suppurative process on his left side, and from the history it appeared that this condition had existed for several years. This finding led me to the conclusion that the condition of his larynx and trachea was secondary to this chronic suppurative sinuitis. The microscopic examination of the tissue removed showed it to be a true hyperplasia of the epithelium as well as of the glandular structures. Such pathologic changes are frequently reported as resulting from suppurative conditions of the nasal accessory sinuses and nasopharynx. I then attempted to do an intranasal operation on his middle turbinate and ethmoid cells, but was very unsuccessful, owing to my inability to obtain sufficient local anesthesia. After keeping the laryngotracheal cleft open for four and one-half months and dilatating the laryngeal chink, we had a very satisfactory result, and he was ready for the plastic closure or laryngostomy (Figure 17). The gap being of considerable width and length, and inasmuch as about one-half of it was of the trachea, I feared that skin flaps from the neighborhood would not suffice, in view of the danger of being sucked in with any effort at inspiration. Consequently, I sought a method of employing some solid organic material, and determined to transplant a section of a rib. It is well known that bone transplants do not do well and become absorbed in locations where there is not normally bony structure; yet I

was hopeful that sufficient osteal or periosteal tissue would remain to form a firm closure. Therefore, under general nitrous oxid oxygen anesthesia by the intratracheal method, aided by scopolamin-morphin, a section of the right eighth rib, two inches long, was removed with its periosteum and put into a subcutaneous tunnel previously prepared in the side of the neck. This healed in fairly well, in spite of some suppuration (Figure 18). After three more weeks, under the same type of general anesthesia, this rib transplant with its overlying skin was again made into a flap with its hinged pedicle on the side of the laryngotracheal cleft. This brought the skin within the interior of the larynx and trachea. The opposite lateral margin of the cleft was now freshened and the flap containing the rib transplant sutured in, thus closing the cleft of the larynx and trachea. The top surface of the flap now was made up of a thick mass of granulations which should have been immediately covered with some sort of skin flap or graft. As a consequence, the granulation shrunk, leaving the bone graft without sufficient nutrition, and this procedure proved to be a failure. Two weeks later I attempted the double transfer method of grafting, by what is known as the "Kausch method"—namely, employing the second toe of left foot, engrafted into palm of right hand and subsequently transferring it into the laryngotracheal cleft. A toe was employed instead of the finger, because he is already handicapped by having lost two of his fingers by accident while at work as a machinist.

While the toe and hand were joined and sutured, they were kept immobilized by encasing the greater portion of the body in a cast. It is best to have the patient practice this position by strapping him in a jacket made of heavy buckram (Figure 19). One must see that the normal functions of the bowels and bladder are possible while in this restrained cast. All went well, although the patient was very uncomfortable, until the ninth day, when, during the night, the patient had a nightmare, and with a violent effort loosened the attachment of hand and foot. When I took off the bandage I found the union to be slight, and since the patient appeared to be much depressed by the failure, I desisted from reattaching the toe and hand and reapplying the cast. This, then, was the second failure. While discussing the case with a general surgeon (Dr.

W.) and planning to employ fascia lata transplant, he suggested that I try a flap containing a part of the clavicle.

One month from the last attempt I operated in the following manner, under scopolamin-morphin and ether anesthesia: A skin flap was made, the pedicle being located in the side of the neck and the periphery over the inner one of the clavicle. Dissecting this later portion as far as the upper and lower border of the clavicle with a large flat chisel, I removed a flat piece of this bone the entire width of the clavicle and one and one-fourth inches long. I now turned the dermal layer of the flap under this bone chip, surrounding it, so to speak, and left it in situ, so as to have it well nourished for the next step. Ten days later I loosened up the entire flap, including the bone, and turned it into the defect so that the dermal layer looked inside of the larynx. Along the side margins of the cleft, the flap was denuded of the epithelium and stitched by a few interrupted sutures into them. The primary defect over the clavicle was immediately sutured. The union of the implant was very satisfactory, and on the eighth day the pedicle was severed and the closure of the cleft perfected, as well as the remaining defect over the clavicle closed. The ultimate result from this last procedure was very satisfactory (Figure 20).

Following all these procedures, the patient left the hospital with a very good breathing tube and a very fair voice. Laryngeal examination revealed almost complete fixation of the arytenoids and a single nodular excrescence above what appeared to be the remains of the vocal cords. There was but slight vibration observed from them. The man now obtained a position as chauffeur and had practically no trouble, except whenever he exerted himself he would experience some difficulty in breathing. I decided upon dilatation by bougies, etc., by means of suspension, but found that I was absolutely unable to do this. This, I believe, was due to the laryngostomy operation, in that the newly made larynx was placed so much more anteriorly that it could not be brought into view, at least not by means of the Albrecht or Killian instruments. Yet I felt sure that I could do so by means of the new Lynch apparatus. I decided to wait for it, and on October 12th I suspended him, and with the aid of the longest spatula, I introduced Ingalls' long flexible tube, of which I took an X-ray while in situ (Figure 21). This tube was permitted to remain

for three and one-half hours, and following its removal the patient declared that he breathed very freely and his voice was better than it had been for a long time. However, within an hour or two he showed signs of edema and extension of inflammation into the bronchi. He appeared to be in imminent danger of pneumonia.

On the fourth day from the time of the above mentioned procedure he breathed so laboriously that I was forced to do a tracheotomy. This was very easily performed in the upper extreme of the tracheal defect, just on the side of the clavicle graft. I found the bone still intact and the trachea and larynx smooth within, and no evidence of any constriction. The suppuration from his nasal accessory sinuses still annoys him considerably, and believing that it was keeping up the laryngeal trouble, I felt that it was advisable to operate thoroughly and to forcibly dilate the larynx, as well as remove the excrescence mentioned above.

On November 8th, three weeks from the time of performing the tracheotomy, I placed him under a general ether vapor anesthesia and suspended and dilated, with considerable ease. I also cleansed out the ethmoid and opened the rest of the nasal accessory sinuses. The suspension and extreme effort to get the much displaced larynx into view caused the spatula to come too forcibly in contact with the lower jaw, and a very disagreeable accident occurred, causing the three central teeth to turn outward and hang merely on the gingival mucous membrane. I replaced them immediately in their sockets and wired them to the neighboring teeth. He recovered from these last procedures, and now is in splendid physical condition, with respiration and voice improved. The nasal accessory sinus suppuration has practically disappeared. He is now wearing this automatic closing tracheotomy tube, so that I may be certain that the space for breathing between the vocal cords is permanently large enough.

On May 10, 1916, I closed the tracheal opening, and the patient is now breathing freely in the natural way and he has a fairly good voice.

In this connection I desire to present this new device which I call an automatic closing tracheotomy tube.

The indication for the use of this instrument is when the patient desires to speak and objects to placing his finger con-

tinually over the opening of the tube, which is conducive to irritation and infection of the wound, and which is always associated with considerable embarrassment.

Particularly is this of value in the treatment of such conditions as bilateral abductor paralysis, or irremovable obstructions below the tracheotomy tube, as from cicatrix, malignant disease, etc.

The instrument consists of a regular tracheotomy tube, the inner tube being very short, having attached to it a shutter which is operated by a flexible rod passing through the flexible cable tube. At the distal end of the apparatus is seen a spring push button, which is attached to the rod, and by compression closes the opening of the tracheotomy tube, the opening of which is again reestablished when the button recoils by spring action (Figure 22).

The entire instrument is hidden from external view, as shown (Figure 23), the button being carried through an opening into the man's trouser pocket, or in the pocket of a woman's skirt. It may pass through the sleeve of the patient, being held hidden in the palm of the hand.

Figures 24 and 25 show the instrument in action.

NOTE.—Since reading this paper I have had considerable additional personal experience and have discussed suspension laryngoscopy with many others, and have watched not a few at work. I have received the impression from all these sources that there is a greater limitation to suspension than was formerly believed. I am, however, even more firmly convinced than heretofore of its great value in the diagnosis and surgical treatment of the larynx and trachea. General anesthesia (nitrous oxid ether vapor) is of great assistance, but local anesthesia is more practical. I have had better success since I have followed the suggestion of Israel of Houston, who recommends the application of pure flaked cocain over the tongue, pharynx and larynx, thereby securing a better anesthesia than by swabbing solutions or injecting the superior laryngeal nerve.

Another valuable method which I desire to place on record is the socalled coagulation method of Percy. A copper rod heated by electricity is applied, the surrounding tissues being protected by ice cooled speculum, etc. Figure 26 shows a modified Lynch speculum in the form of a sheath, into which fits

the cooling speculum or tube. The cooling process is accomplished by permitting ice water to flow through its double wall. In addition, there is passed a metal flexible tube containing two smaller tubes through which cold water flows into the upper part of the esophagus, preventing scorching of that organ. In addition, two plates resembling Leiter's coils are placed on the sides of the larynx to prevent scorching of the neck tissues. I am also using Lynch's modeling compound aluminum plates, which do away with some of the difficulties mentioned in the paper.

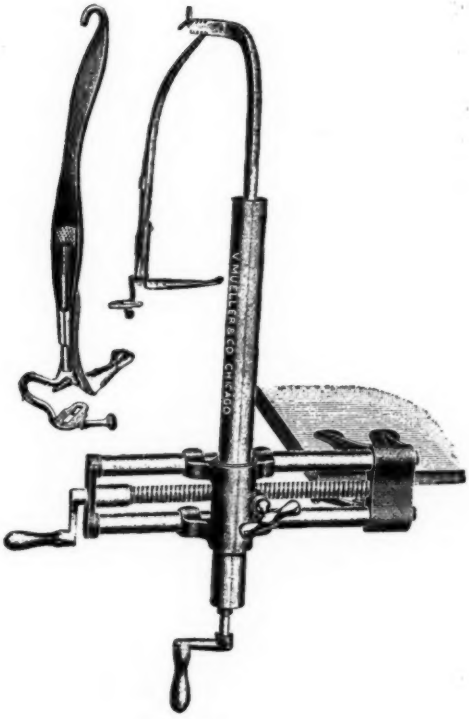

FIGURE 1.

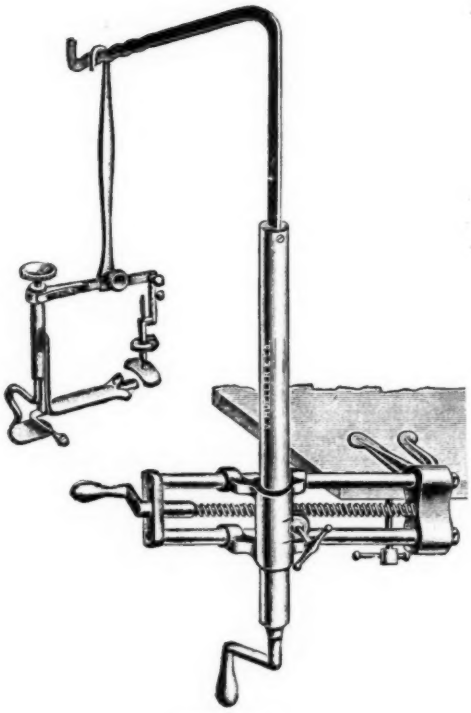

FIGURE 2.

Fig. 1

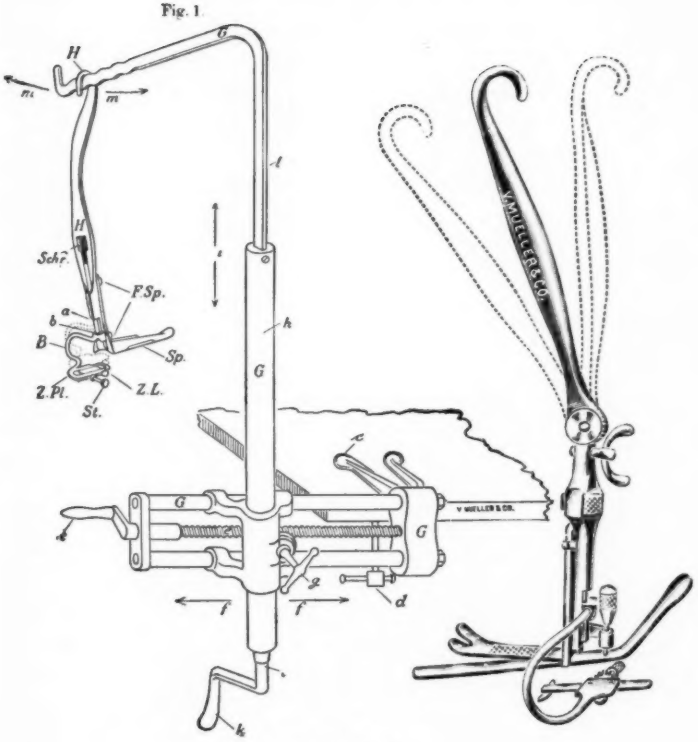

FIGURE 3.

FIGURE 4.

FIGURE 5.

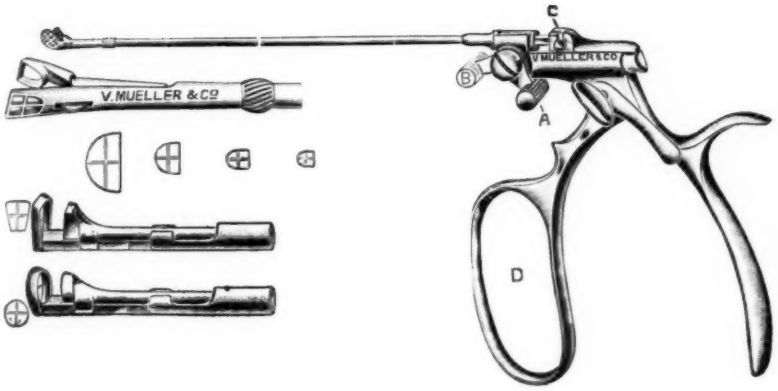

FIGURE 7.

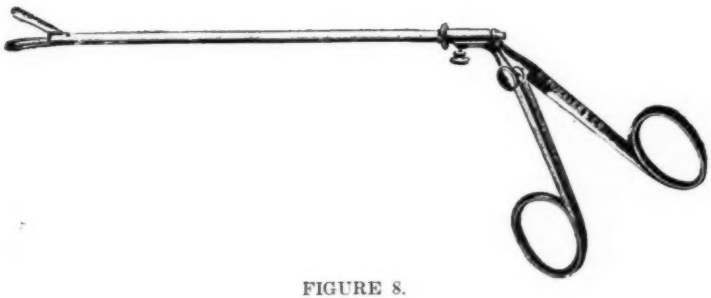

FIGURE 8.

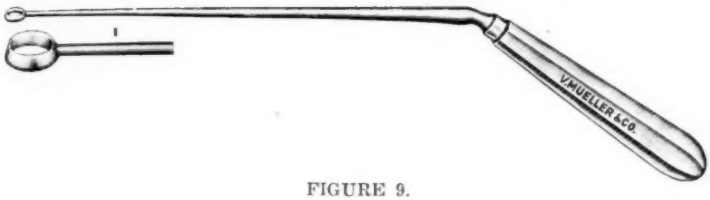

FIGURE 9.

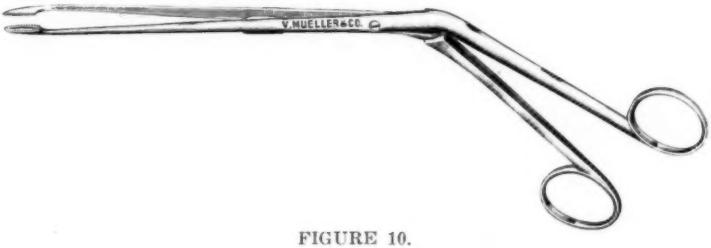

FIGURE 10.

FIGURE 11.

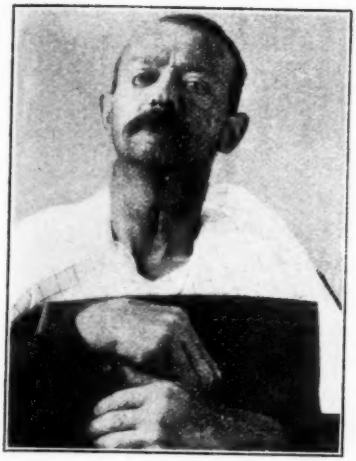

FIGURE 12.

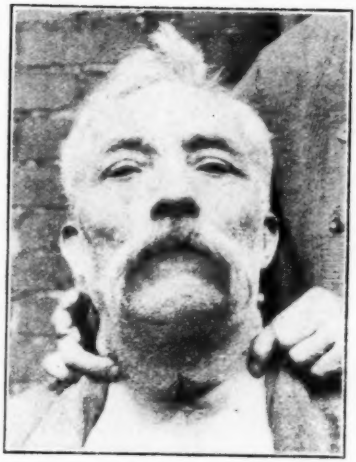

FIGURE 14.

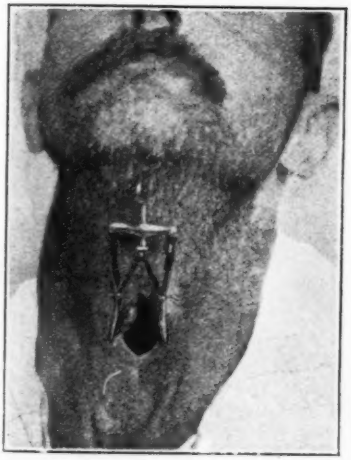

FIGURE 13.

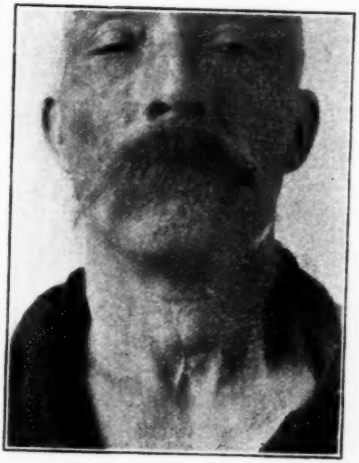

FIGURE 15

FIGURE 16.

FIGURE 17.

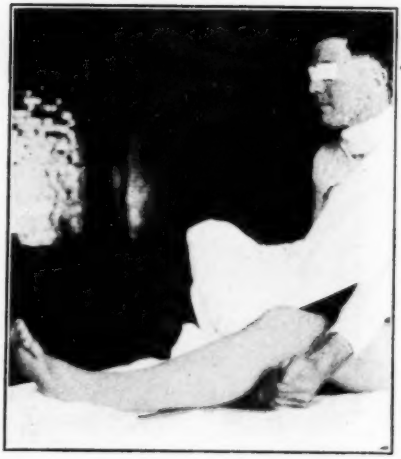

FIGURE 19.

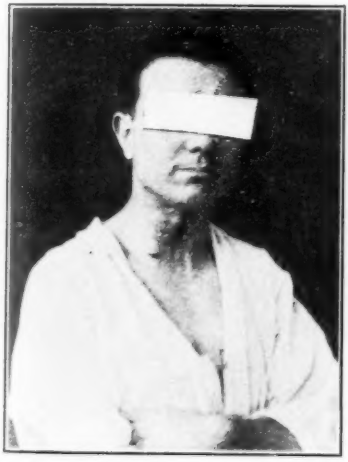

FIGURE 18.

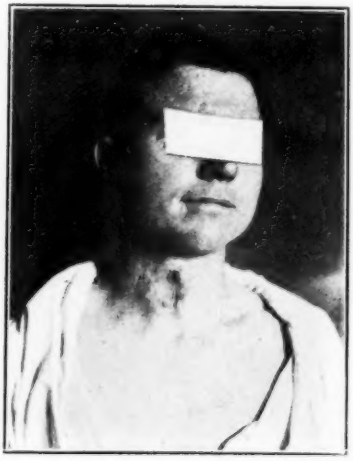

FIGURE 20.

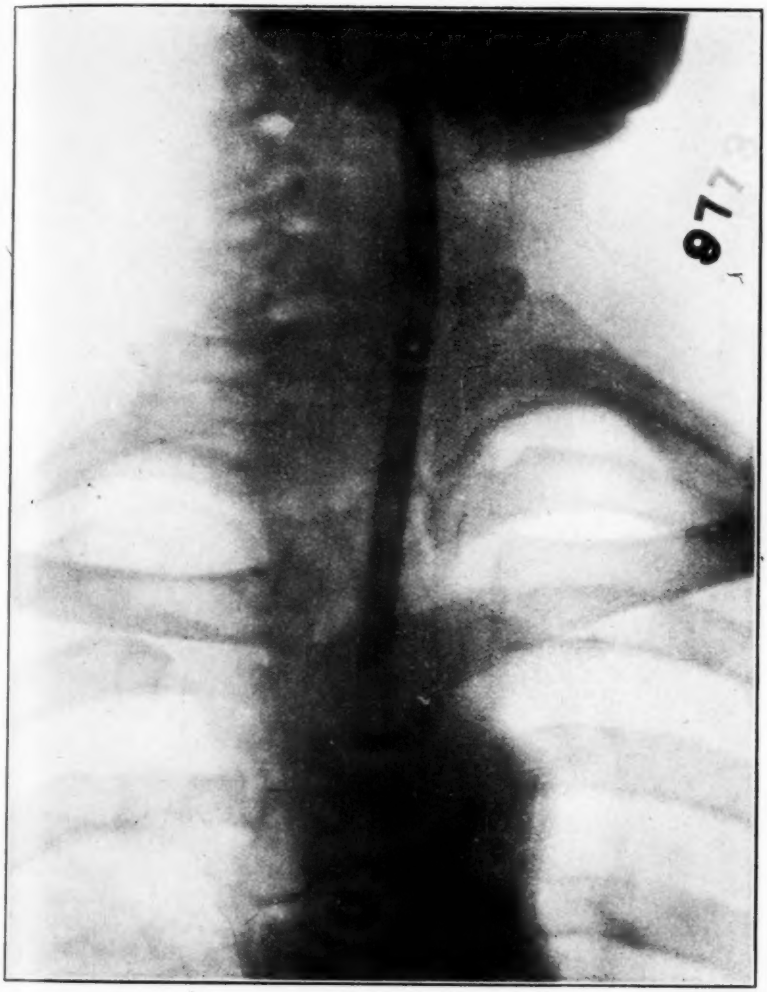

FIGURE 21.

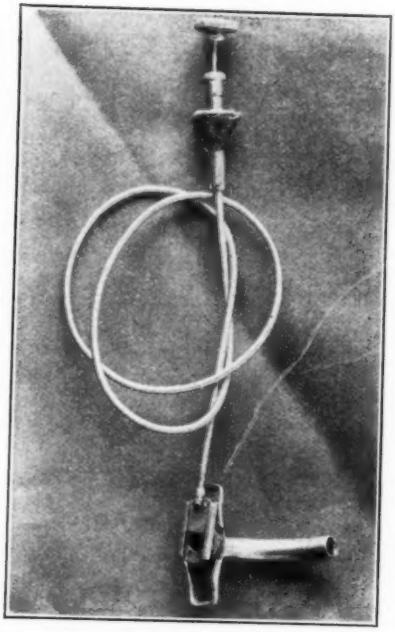

FIGURE 22.

FIGURE 23.

FIGURE 25.

FIGURE 24.

FIGURE 26.

XII.

THE GRAM STAIN IN MAKING A BACTERIOLOGIC DIAGNOSIS IN OTOLARYNGOLOGY.

By A. B. Bennett, M. D.,

Washington, D. C.

When vaccin therapy was first suggested in ear, nose and throat practice, I thought it an impractical idea unless associated with elaborate laboratory work, owing to the extensive bacterial flora found usually about the mouth and throat. Later on, Dr. E. R. Stitt, U. S. N., suggested that I try a routine Gram stain on the secretions of ear, nose and throat cases, and determine if it would not prove a valuable aid in making a bacteriologic diagnosis. I have followed the suggestion, and take pleasure in presenting a synopsis of this work on over one hundred and sixty cases, which includes a microscopic study of over three hundred Gram stained smears.

The Gram stain is especially adapted for a rapid determination of the bacterial contents of secretions from the ear, nose and throat. It takes but a few minutes to prepare the specimen for the microscope, and it differentiates to a great degree of accuracy the organisms found. Gram positive bacteria are those which retain the gentian violet after decolorizing and counterstaining, and Gram negative bacteria are those which do decolorize and take on counterstain.

The bacteria which are Gram positive are the staphylococcus aureus, albus, and pyogenes, streptococcus, M. tetragenus, pneumococcus, anthrax bacillus, tubercle bacillus, lepra bacillus, tetanus bacillus, diphtheria bacillus, diphtheroid bacillus, B. aerogenes capsulatus, odium albicans and mycelium of actinomyces, saccharomyces, Hoffman's bacillus, and B. xerosis.

The Gram positive bacteria which may be considered important from an ear, nose and throat standpoint are the staphylococcus, streptococcus, M. tetragenus, pneumococcus, tubercle bacillus, and diphtheria and diphtheroid bacillus. Each of

these can be recognized from its characteristic morphology. The microscope cannot differentiate between the staphylococcus aureus and albus, but such differentiation is not important. The recognition of the tubercle bacillus would have to be confirmed by the acid fast test, and the B. diphtheria cannot be distinguished from the B. diphtheroid, as both have a changing morphology from dotted to oblong segments, and, according to some eminent bacteriologists, even the guinea pig test is not absolute as a point of differentiation.

The Gram negative bacteria are the meningococcus, M. catarrhalis, M. melitensis, B. typhosus, B. coli communis, B. dysenteriæ (Shiga), spirillum choleræ asiaticæ, B. pyocyaneus, B. mallei, B. pneumoniæ, Friedlander group, B. proteus, B. influenzæ, B. of bubonic plague, B. of chancroid, B. of Koch-Weeks, and the gonococcus.

The Gram negative bacteria which may be considered as important from an ear, nose and throat standpoint are the M. catarrhalis, B. pyocyaneus, the Friedlander group and B. of influenza. Here again recognition becomes fairly easy, as the only Gram negative coccus is the M. catarrhalis. The B. pyocyaneus, while capable of many forms, is usually seen as a rod shaped organism very similar to the typhoid or colon bacillus, which distinguishes it under the microscope from the Friedlander group, which show a distinct capsule. No bacteriologist will admit the absolute diagnosis of an organism until it has been cultured and tested.

In the early part of this work I had all the secretions cultured, also occasionally throughout the year, and the smear diagnosis and the culture diagnosis have agreed with unfailing constancy. However, it often happened that in a mixed infection the culture would show a different predominating organism than the smear, due to the fact that the predominating organism in the discharge was a poor grower on artificial media and was outgrown by a less numerous organism which happened to be a good grower on artificial media. In many laboratories a smear is considered an essential control of a culture in order to make sure that the predominating organism in the pus has not been outgrown by an unimportant organism which is in the pus or has been accidentally introduced. Another important condition shown by the stained smear is phagocytosis, which is an excellent factor in deter-

mining the active infectious organism when confronted with a mixed infection, and it also indicates the resistance of the patient against the organism.

I have grouped this series of about one hundred and sixty cases by diseases, and will briefly summarize the findings under each head.

DISEASES OF THE NOSE AND THROAT.

There were seventy-six cases of acute catarrhal sinuitis, one or more cavities involved, and of different degrees of intensity, but all showing on irrigation of the nose the characteristic catarrhal sinus secretion. The smears were all made from these secretions, taken directly from the nose with no possibility of mouth contamination.

In twenty cases the first smear showed no organism, but five on subsequent examinations showed the presence of bacteria, and nine remained free from microscopic evidence of infection, indicating that probably such a condition as a purely noninfectious acute catarrhal sinuitis does occur. In six there were no subsequent examinations made. These cases made very prompt recoveries under simple local treatment.

In forty-two cases (fifty-seven and one-half per cent of the total number) there was a single or pure infection—twenty-two with the pneumococcus, fourteen with the staphylococcus, and six with the M. catarrhalis.

In fourteen cases there was a mixed infection—five had the pneumococcus predominating and phagocytized, two had the staphylococcus, one had the Friedlander group, and one the streptococcus predominating and phagocytized.

Six cases showed the bacteria in about equal numbers—the diphtheria bacillus and the staphylococcus in one, the pneumocuccus and the staphylococcus in four, the M. catarrhalis and staphylococcus in one.

In one case the first smear showed a pure staphylococcus, and one month later the secretions showed only the pneumococcus and the M. tetragenus.

Many of these cases were clinically la grippe or epidemic influenza; four cases due to a pure pneumococcus infection were from one house epidemic; four cases due to a pure staphylococcus infection came from two house epidemics, two cases coming from each house. The main points in this series

is that the great majority of the cases of acute catarrhal sinuitis are infectious, that over fifty-five per cent were due to a single or pure infection, and that the pneumococcus and the staphylococcus were the most frequent causes.

There were ten cases of chronic catarrhal sinuitis involving one or more cavities, having existed for long periods of time and exhibiting on nasal irrigation the characteristic catarrhal thick yellow or yellowish green heavy secretions. The smears and cultures were made directly from these secretions, without permitting any mouth contamination.

In two cases there was a pure staphylococcus infection, and one case of mixed infection, where the staphylococcus predominated and was phagocytized, although the pneumococus, M. catarrhalis, Friedlander group, B. pyocyaneus and the M. tetragenus were also present. Another case had the same multiple infection without any one organism predominating.

In two cases the B. pyocyaneus predominated, with the pneumococcus and M. catarrhalis present.

Two cases had only the pneumococcus and M. catarrhalis, and another only the pneumococcus and Friedlander present. The last of these cases I will report in greater detail, as follows:

A young man, with a history of chronic catarrhal sinuitis of three years' duration, accompanied by hoarseness and severe cough and expectoration, occasional night sweats and no loss of weight. There was a tumor on the left thyroid cartilage, obscuring the left vocal cord. The first smear from the sinus secretions had the pneumococcus predominating and phagocytized with the M. catarrhalis; streptococcus and staphylococcus also present. The sputum smear proved a pure pneumococcus infection markedly phagocytized. I gave him two injections of pneumovaccin, which was followed by a decided improvement and a practical disappearance of sinus symptoms. I also made a smear from a swab applied directly to the laryngeal tumor, and it contained only pneumococci, and no tubercle bacilli. I gave him deep inhalations of medicated oil, which lessened the cough and expectoration, with no change in the tumor. I again examined the sputum and found abundant tubercle bacilli with only a few pneumococci. I continued the oil inhalations for several weeks, the patient improved generally—gained weight, and the cough and the expectorations

practically ceased, the last sputum examinations showing no tubercle bacilli and but a few pneumococci. The laryngeal conditions remained the same; the patient then moved to another part of the country. In this case the active pneumococcus infection obscured the tubercle infection, which was revealed on relief of the acute inflammation. In these cases of chronic catarrhal sinuitis a multiple infection seems the rule, and the staphylococcus, and the B. pyocyaneus are the most frequent offenders.

There was only one case of acute suppurative sinuitis, and that occurred in the right maxillary sinus. Pulsating pus was flowing into the middle meatus, and other cardinal symptoms were present. The smears and culture showed a pure staphylococci infection. I gave five injections of an autogenous vaccin along with local treatment, and obtained a quick recovery.

There were nine cases of chronic suppurative sinuitis with free pus flowing from the infected cavities. Two were pure infection, one being pneumococcic and the other B. pyocyaneus. Two were of dental origin—in one of these the smear exhibited a pure staphylococcus infection, and the other a mixed infection of M. catarrhalis, B. pyocyaneus, B. diphtheriæ, pneumococcus, staphylococcus, and streptococcus. In one case there was also caries of the ethmoid bone, and the smear showed a mixed infection, the streptococcus predominating and phagocytized with the M. catarrhalis, Friedlander group, pneumococcus and the fusiform bacillus also present. Another case contained staphylococci, streptococci, pneumococci, and B. pyocyaneus, and another case contained pneumococci, staphylococci, streptococci, M. catarrhalis and the B. influenzæ. In the last of these cases the first smear revealed a pure streptococci infection with marked phagocytosis. Later there was, in addition to the streptococci, a few staphylococci; four months later the infection appeared principally staphylococcic with a few streptococci, and a few weeks later the streptococci and staphylococci were in equal amount and there appeared a few B. pyocyaneus, the culture showing staphylococci and streptococci, each forty per cent, and the bacillus pyocyaneus, twenty per cent.

There were sixteen cases of chronic nasopharyngitis. The smear from one contained only an epithelial débris with no organisms. The others were all mixed infections, with pneu-

mococcus predominating in nine, and the bacillus pyocyaneus being very numerous in nine. The other organisms found were the staphylococci, streptococci, Friedlander group, M. catarrhalis, diphtheroid bacillus and M. tetragenus. One of the features of the smears was the presence of large epithelial cells. Some of these were specimens which were claimed to come from other sources, but the picture was typical of a nasopharyngitis. In five cases I used mixed vaccin as indicated by the microscope, and in four there was decided benefit.

There was one case of acute nasopharyngitis. The smear revealed a pure staphylococci infection, and a culture contained a pure growth of the staphylococcus albus. Later the inflammation extended to the sinuses, and the smears from the sinus secretions contained staphylococci and pneumococci.

There were five cases of atrophic rhinitis. In three the smear contained a mixed infection of diphtheroid bacillus, staphylococci, M. catarrhalis, M. tetragenus, pneumococci and B. pyocyaneus. In the fourth case the smear contained only the diphtheroid bacillus and B. pyocyaneus. In two cases I used an autogenous vaccine of all the organisms found in the discharge. One case received thirteen injections, with slight improvement. The other case I have given over thirty-one injections, the dose increasing to 2,000 million; the local improvement was marked and the improvement in general health was quite remarkable.

A smear from an ulcer of the nasal septum contained principally a fibrous exudate, with only occasionally a single pneumococcus and a staphylococcus—really no infection. This ulcer proved to be due to syphilis that had been contracted thirty years before.

One patient complained of a nocturnal accumulation of pus in his throat and mouth. His nose, throat and larynx were normal. A specimen of pus brought by the patient corresponded exactly with the pus expressed from suppurating gums about two molar teeth, and contained pneumococci, M. catarrhalis, bacillus pyocyaneus, Friedlander group, leptothrix and spirocheta.

In one case of acute tonsillitis, where a thin membrane had extended over the posterior pillar, I peeled off a strip of the membrane and the smear contained pneumococci predominating with streptococci, staphylococci, M. tetragenus, M. catarrhalis, and a long search revealed no diphtheria bacilli.

In examining sputa from the larynx, trachea and bronchi, I always washed the secretions in sterile water, to remove the saliva and its possible bacteria before smearing it on the slide. There were two cases of acute laryngotracheitis. In one there were no organisms, and in the other there was a mixed infection, the staphylococci, pneumococci, diphtheria bacillus, M. tetragenus, M. catarrhalis, Friedlander group, and B. pyocyaneus being present. In this case I gave a mixed vaccin with good result.

One case of chronic tracheitis contained in the smear the pneumococcus predominating and phagocytized with the M. tetragenus, M. catarrhalis, staphylococci and B. pyocyaneus also present. I gave the patient four injections of pneumococcus vaccin with a good result.

One case of chronic laryngitis due to habitual clearing of the throat was interesting in that I examined the sputum many times and always found no organisms present.

In four cases of chronic tracheobronchitis the smears all indicated mixed infections of pneumococci, staphylococci, streptococci, M. tetragenus, Friedlander group, B. pyocyaneus, and M. catarrhalis. I gave a mixed vaccin to two of these cases, and though they had been of long standing there was a prompt response and recovery.

There were two cases of laryngeal carcinoma, in each of which the smear contained all the organisms found about the mouth and throat, with numerous pus cells but no phagocytosis.

I have examined the pus from a great number of cases of chronic follicular tonsillitis, and the microscopic picture was invariably the same, showing a mass of different organisms, among them being the pneumococcus, staphylococcus, streptococcus, M. tetragenus, M. catarrhalis, diphtheroid bacillus, B. pyocyaneus, Friedlander group, leptothrix, spirocheta, and fusiform bacillus.

DISEASES OF THE EAR.

In cases with aural discharge the microscopic examination is much more satisfactory than with discharge from the nose and throat, for the reason that contamination is practically impossible. Of course, if the smear is made from pus lying in the auditory canal, there is a possibility of other organisms being present from the skin about the canal. But if the canal

is first wiped dry, the ear inflated, and the pus which flows from the middle ear through the perforation is caught up on a sterile cotton swab and a smear made, there is certainly very little chance of other than the organism really in the discharge being present.

There were five cases of acute suppurative otitis media. In two the infection was a pure staphylococci infection, one was a pneumococcus, and one was a pure infection of the diphtheria bacillus; and the last case was very acute, having violent pain, rupture of the membrana tympani, and an outflow of serosanguineous fluid, a smear from which failed to reveal any organisms—this case ceased to discharge on the second day. The first two cases also made rapid recoveries under simple local treatment.

There were six cases of acute suppurative otitis media with mastoiditis. Three cases showed a pure pneumococcus infection, two made complete recoveries under the use of pneumovaccin and local treatment, and one was operated upon. Another case was of a few weeks' duration, the external plate of the mastoid had ruptured, and there was fluctuating pus under the integument. The smear contained a tremendous number of streptococci and fusiform bacilli. I did a mastoid operation immediately, and in two weeks made a smear from the wound, and there were no organisms present. In the fourth case the conditions came on as a complication during an attack of measles. The smear showed a pure streptococcic infection, with a high degree of phagocytosis. The patient was desperately ill. I used streptococcus vaccin with decided benefit, followed by recovery. There was an apparent closure of the perforation and hearing power for the watch of 16/30. The perforation evidently opened, for one year later the child's mother dropped into this ear some "sterile" boric acid solution, followed by an acute suppuration, the smear indicating a pure diphtheroid infection. There was a quick recovery under simple local treatment. Four months after this attack the patient was bathing in a swimming pool and there was again an acute suppuration from that ear, the smear showing a pure staphylococcic infection. Simple local treatment again produced a quick recovery. In another case the M. catarrhalis was the infectious agent, and the case went to operation. It is interesting to note that in the ten cases of acute sup-

purative otitis media where an infection was found, it was always a pure infection, and that in one case with three different attacks, it was a pure infection of a different organism each time, and not the breaking out of the first infection, as one might readily suppose.

There were sixteen cases of chronic suppurative otitis media. The suppurations had been of six months' to thirty years' duration. In two cases there was a pure diphtheroid infection. In three cases there was a pure staphylococcus infection, in one of which there was a marked phagocytosis; in another there was also a Gram positive fungus present; and in the third there was a prompt cessation of discharge after using vaccin; later the discharge reappeared and was different in character than before, the smears and culture showing a pure infection with the B. pyocyaneus. In four cases the B. pyocyaneus was the active cause. One of these cases was of thirty years' duration; both the smears and culture showed a pure infection, and I gave eleven injections of autogenous vaccin, followed by complete recovery. Another case, also of thirty years' duration, contained on the smear a few diphtheroid bacilli and staphylococci, with the B. pyocyaneus predominating—the culture was pure B. pyocyaneus. I administered eleven doses of an autogenous vaccin, and the ear has been absolutely dry for eighteen months. The third and fourth cases merely showed the B. pyocyaneus as predominating, with a few diphtheroid bacilli present. In two cases the M. catarrhalis was the only organism found in the pus taken from the middle ear; however, in one of these the first smear of pus taken from the canal showed a few diphtheroid bacilli with the M. catarrhalis. Both of these cases made early recoveries under local treatment. There were three cases of mixed infection, two with the B. pyocyaneus, diphtheroid bacilli, staphylococci, and the other with B. pyocyaneus, diphtheroid bacilli, M. catarrhalis, and M. tetragenus.

If I may take the time, I would like to report a case in detail:

W. D. F., male, white, married, aged thirty-one years. March 29, 1912. Diagnosis: Chronic suppurative otitis media, right; chronic suppurative otitis media, residual, left. Duration: Intermittently for twenty-nine years. Membrana tympani, right, absent; membrana tympani, left, scarred and adhered to promontory. Smears and culture from right ear

indicated a pure B. diphtheriæ infection. Administered seven increasing doses of autogenous vaccin. The discharge ceased shortly after beginning treatment, but I continued the vaccin, hoping to establish an immunity from a recurrence.

October 18th, six months later, the patient reported, with right ear still dry, but having a discharge from the left ear. A smear revealed pure bacillus diphtheriæ infection. I used all manner of antiseptics in the ear, including solutions of the silver salts and of iodin, until November 14th, during which time frequent smears showed a steadily increasing number of diphtheria bacilli—the last smear showing a solid mass of the organisms. I then blew into the ear some acetanilid and boric acid powder, which immediately stopped all discharge.

On December 19th, one month later, the right ear recommenced to discharge, accompanied by pain and throbbing; the discharge contained bacillus diphtheriæ and staphylococci in equal number, and both phagocytized. The left ear had also started slightly, and a smear from that side contained a few staphylococci.

On January 4th he reported, with right ear discharging, and a smear contained a large amount of B. diphtheriæ. He also was suffering with an attack of acute sinuitis, and a smear from the sinus secretions contained B. diphtheriæ and staphylococci. Five days later he was practically well and the ears were dry. I then examined the pus from his tonsil crypts, and found the leptothrix, B. pyocyaneus, M. catarrhalis, staphylococci, streptococci, pneumococci and a few B. diphtheriæ.

On January 22nd he reported, with a severe cold and the right ear discharging freely. The smears contained a few staphylococci and B. diphtheriæ, both phagocytized. I gave him three doses of a stock staphylococci vaccin, and on February 3rd the right ear was dry, but the left ear was moist, a smear showing a pure staphylococcus infection. He then passed from observation.

In this case there was no immunity produced by the diphtheria vaccin to a reinfection by the B. diphtheriæ, which was to be expected. The best fluid antiseptics were of no avail, while powders blown into the ear proved of great value; and lastly, the three doses of staphylococcus vaccin had not made much impression, as the left ear was being infected with the staphylococcus at the last observation.

There were five cases of chronic suppurative otitis media with mastoiditis. The suppurations had existed for a long period of time, and there was occasional pain and tenderness over the mastoid process. One case was in a negro baby, a neglected case, and the smear indicated a streptococcic infection, with the staphylococcus, M. catarrhalis and the fusiform bacillus also present. The second case, of twenty-six years' duration, was a pure infection of the diphtheroid bacillus, and was quickly cured by simple local measures. The third case was of twenty years' duration, and the smear contained apparently a pure culture of B. pyocyaneus. The culture contained two organisms, one being an immobile pyocyaneus bacillus which agglutinated in the presence of the patient's blood. An autogenous vaccin was made and four doses given, with complete recovery. In the fourth case the suppurations had existed since an attack of typhoid fever in Porto Rico in 1898. The smear and culture contained about ten different organisms, one of which was culturally the B. typhoid. It was proven to be the typhoid bacilli, and it agglutinated in the presence of the patient's blood. I gave him typhoid vaccin, and there was a decided improvement. This case was an aural typhoid carrier. He passed from observation before I could make any further tests. The fifth case was of seven years' duration, the smears showed great numbers of staphylococci phagocytized and a few diphtheroids. The sixth case I will report in brief detail:

October 18, 1912. A. S. B., male, white, single, aged fifteen years. Chronic suppurative otitis media of ten years' duration. Mastoid process tender and often painful. Membrana tympani absent, and pus flowing from posterior superior quadrant of middle ear. First smear and culture contain a pure culture of B. pyocyaneus. Administered three doses of stock pyocyaneus vaccin, and second smear contained less B. pyocyaneus and a few M. catarrhalis and staphylococci. Gave four doses of stock pyocyaneus vaccin, and the third smear contained no B. pyocyaneus, and a few staphylococci phagocytized. On December 15th the fourth smear contained a few staphylococci and no other organisms. On December 31st the fifth smear contained the B. pyocyaneus and staphylococci. Administered two large doses of pyocyaneus vaccin, and on January 14th the sixth smear contained a marked in-

crease in number of B. pyocyaneus, and culturally there were the B. pyocyaneus and staphylococci. Administered six doses of autogenous vaccin, and on February 14th the seventh smear contained a large number of the B. pyocyaneus and staphylococci. I gave him six more doses of autogenous vaccin, and on March 27th the discharge was much less, and the eighth smear showed no B. pyocyaneus and only a few staphylococci. I continued giving the autogenous vaccin until twelve more doses were given, and the ninth smear contained the B. pyocyaneus in great numbers and a few staphylococci. The discharge is much less than originally and the boy's general health is better, but in spite of eight doses of stock pyocyaneus vaccin and twenty-four doses of autogenous vaccin, of the B. pyocyaneus and staphylococcus mixed, the pus from the ear still contains B. pyocyaneus and staphylococci.

There was one case of mastoiditis unaccompanied by any middle ear symptoms at the time of my first examination. Four weeks before I saw her there had been an acute middle ear suppuration which entirely recovered, and the membrana tympani and hearing were normal. Ten days before I saw her a swelling appeared over the mastoid that had steadily increased, and at my first examination there was a large fluctuating mass over the mastoid process. The child's health was good, appetite normal, and there had been no fever for the past week. I performed a simple mastoid operation. A smear from the pus that flowed from the first incision contained a very few staphylococci, all phagocytized, and no other organism, which agreed perfectly with the clinical picture, that she had entirely overcome the infection and that the pus and débris of the infection were lying under the skin.

In these twenty-four cases of chronic suppurative otitis media the B. pyocyaneus was the chief offender, with the staphylococcus and B. diphtheroid running close seconds. Twelve were pure infections, and ten were mixed infections.

This report is made entirely from the record cards in my own practice, and it has taken some time to accumulate even this small amount of data. The lesson has been invaluable to me, and I am sincerely enthusiastic over this metrod of acquiring an additional fact in the diagnosis of my cases. If I may

draw conclusions from the smears I have made and personally studied, and from the cultures that I have taken and which have been grown, plated and differentiated in the laboratories of the U. S. Naval Medical School and the National Vaccin and Antitoxin Institute, they are these:

1. The active organisms infecting the ear, nose and throat are the staphylococcus albus and aureus, streptococcus, M. catarrhalis, M. tetragenus, pneumococcus, B. pyocyaneus, B. influenzæ, Friedlander group, and the diphtheria and diphtheroid bacilli. Most of these can be recognized under the microscope by the Gram method of staining. The fusiform bacillus is readily recognized, also, and is rarely seen except when there is decay or caries present.

2. The spirocheta and leptothrix seem to be inhibited, or at least inactive, in these diseased conditions. When the spirocheta are associated with fusiform bacilli, with the absence of other organisms, the picture is of Vincent's angina.

3. The Gram stained smear is of great value when administering vaccins, because, in the majority of instances, there is either a pure infection readily recognized, or there is a predominance and phagocytosis of one organism to indicate the vaccin needed; also because the effect of vaccin on the bacterial contents of the discharge can be periodically observed.

4. The Gram stained smear is of great value throughout the course of any infection of the nose, throat and ear, to observe the lessening or increasing of the bacteria, and to detect a change of infection from one organism to another.

5. Lastly and most important is the conclusion that a study of the smear with a recognition of the type of infection gives a knowledge of the prognosis otherwise unattainable. For instance, an infection with the M. catarrhalis, even if it be chronic, is usually generally relieved by simple measures. An acute infection by the pneumococcus is easily relieved and responds well to vaccin therapy. Chronic infections due to the staphylococcus or B. pyocyaneus are less quickly relieved, often taking considerable time and patience. The staphylococci infections are materially affected by vaccin, while the results in using the pyocyaneus vaccin are less certain. The presence in the smear of pus of the fusiform bacillus usually indicates

a grave condition with decay or caries. As is well known, the fusiform bacillus can only be found by the smear, as it does not grow on artificial media. It is impossible to state in detail the various points in a smear that gives one a prognostic vision, but experience with the microscopic pictures, associated with the after-results of the disease, has, I am sure, placed me on the threshold of another knowledge about my cases. I can heartily recommend it as a routine procedure.

XIII.

AN IMPROVED METHOD FOR DRAINING THE TYMPANIC CAVITY IN PURULENT OTITIS MEDIA.*

By John Guttman, M. D.,

New York.

A great majority of diseases of the ear begin with an acute affection of the middle ear. Prompt and proper attention to this middle ear trouble will result in a cure in a very short time; but if this be neglected, it will lead to a functional disturbance, or may result in an intracranial complication, which may become dangerous to life. These facts make it imperative that we pay at least as much attention, if not more, to the acute affections of the middle ear as we do to rarer and less important diseases of the other parts of the ear.

While the etiology and pathology of the most of the middle ear affections, especially those of an inflammatory character, are fairly well known, the therapeutic measures applied in these affections are identical with those in vogue about fifty years ago. In acute catarrhal, or purulent inflammations of the middle ear, we perform a myringotomy with a lancet-shaped needle or with a small knife with a somewhat broader blade, usually without anesthesia, and only in exceptional cases with local or general anesthesia. We thrust the point of a lancet needle into the lower anterior or posterior quadrant, or into the most prominent or bulging part of the drum membrane. The slit in the drum membrane is only as broad as the thickness of the piercing needle. The length of the incision varies. It is through this opening that the viscid or creamy purulent secretion must pass. If the process is very acute, the force of the accumulated secretion widens somewhat the opening, and in the course of weeks, months or years the secretion is discharged through this opening. This surely is no ideal form of drain-

*Read before the Section on Otology, New York Academy of Medicine, January 14, 1916.

age. No surgeon would consider this form of evacuation of pus from a closed cavity satisfactory. The narrow slit in the drum membrane made by the very thin needle or knife will in some cases close in a few hours, either on account of agglutination of some blood, or because some viscid secretion will fill out the small opening of the drum membrane.

To improve the drainage, which is the main therapeutic measure in all acute or subacute cases of purulent or catarrhal inflammations of the middle ear, I devised this little instrument.

This is a hollow steel barrel eight centimeters long, whose upper part is surrounded by a somewhat thicker steel mantel, with long furrows for the purpose of having a stronger and rougher grip in turning the trephine. At the lowest part there is a very sharp circular knife two millimeters in diameter. It is made by Meyrowitz.

This trephine is placed in the lower posterior quadrant, irrespective of the bulging part of the drum membrane, as the drainage will always be better the lower the opening is in the drum membrane. If the drainage is good the bulging part of the drum membrane will disappear spontaneously in three or four days. With two or three turns of the trephine a hole is pierced through the drum membrane. If the trephine is very sharp, a piece of the drum membrane will be found in the hole of the trephine. Immediately after the opening of the drum membrane in cases of otitis media chronica, a pulsating viscid, colorless secretion will make its appearance through the wide opening, and the subjective relief of the patient becomes very marked. In many cases the patient will remark voluntarily: "Now my head feels much easier." The eustachian tube is then catheterized, in order to evacuate the pus from the tympanic cavity.

Up to the present time I employed trephining of the drum membrane in more than thirty cases, in most of them with excellent results. In the beginning, when my technic was not as good as it is at the present time, the results of the operation were not quite satisfactory. I will not trouble you with the details of my statistics.

In looking over the literature I found that Himley advocated the use of a trephine about one hundred years ago; not for the purpose of facilitating the drainage, but to improve

hearing. As it failed in its purpose, and in some cases even caused death, probably because it was handled too roughly, the use of the trephine was given up, as that of a dangerous instrument. In the *Laryngoscope* of 1912, Robert Lewis advocates the use of a punch forceps to remove a portion of the drum membrane, in order to overcome the inadequacy of drainage which sometimes follows ordinary myringotomy in acute cases of otitis media. But inasmuch as he does not describe the punch forceps, nor the method of its use, whether he grasps the drum membrane at the edge of the perforation, or whether he gets a fold of the drum membrane in the grasp of the forceps, whether he uses anesthesia or not, the writer is unable to pass judgment upon this method of procedure.

Although it is not expected that trephining will in all cases replace the present form of myringotomy (by paracentesis) with a lancet-shaped knife, especially in cases where general anesthesia cannot be used, still in numerous well adapted cases of ear affections, especially in subacute or chronic purulent otitis media, where the drainage is insufficient, its superiority to the present form of paracenthesis of the drum membrane will be acknowledged.

XIV.

SARCOMA OF THE NOSE: REPORT OF A FATAL CASE WITH METASTASES IN THE CERVICAL GLANDS AND IN THE BRAIN.*

By J. Payson Clark, M. D.,

Boston.

The patient, Mrs. X., aged sixty-four years, was first seen by me on March 15, 1915, being referred by her physician, Dr. R. L. Toppan, of Newburyport. Three or four months ago she noticed a swelling about the size of a pea on the left side of her nose. It is now (March 15th) about the size of a small marble. It has not been painful at any time. About the middle of February she began to notice a little bleeding on blowing the nose. For the past week this symptom has been more marked and there has been a slight bloody discharge on her pillow in the morning. There is no specific history, and the patient has always enjoyed good health. Examination shows a small rounded tumor, over which the skin is freely movable, on the left side of the nose, just below the nasal bone. On the inside of the left nostril can be seen a dark, purplish tumor on the outer wall just above the vestibule and extending anteriorly to the cartilaginous septum, on which it encroaches slightly. The antra and frontal sinuses transilluminate equally and well. A small piece of the rather firm growth was removed for microscopic examination, the patient's consent to an operation having been first obtained, in case the histologic examination should show the growth to be malignant. The pathologist's report was as follows:

"March 20, 1915. The specimen consists of a small segment of tissue, arising from which is a round white mass measuring about three millimeters in diameter. Microscopically the tissue is composed almost entirely of irregular masses of epithelial cells, which are separated from one another by a connective

*Read at the Annual Meeting of the American Laryngological Association, May, 1916.

tissue stroma. Along one border (surface) there is an inflammatory exudate. The cells in themselves, though entirely of the squamous type, vary in size and shape; but more particularly the nuclei in many cases contain mitotic figures. Opinion: The tumor is a squamous cell cancer.

"(Signed) FRANCIS L. BURNETT, M. D."

March 24th. An operation was performed, under ether anesthesia, with the assistance of Dr. George O. Clark. A longitudinal incision about two and a half centimeters long was made through the skin of the nose in the median line. At the lower end the incision was continued to the left cheek in the depression above the ala. The skin flap was elevated and the growth was seen protruding between the lateral cartilage and the nasal bone. The tumor was then removed with a margin of healthy tissue. The nose was packed lightly with gauze and the skin sutured with horse hair. No external dressing. The wound healed uneventfully by first intention. There was some sinking of the left side of the nose, but the deformity was very slight, considering the amount of tissue which had been removed. There was a small perforation of the septum. Professor F. B. Mallory of the Harvard Medical School kindly examined the growth for me and made the following report:

"Microscopic examination of your tumor of the nose shows that it is rapidly growing and infiltrating, as was obvious in gross. The diagnosis is not so positive. There is no sign of differentiation on the part of the cells. It can be classified either as a carcinoma or possibly as a melanosarcoma, with little evidence of the formation of pigment. It would require the tissue from the primary operation to make a positive diagnosis. I only mention the possibility, because if it were sarcoma, metastases are much more likely to occur. Possibly Dr. Burnett has the sections which he made originally, and I am sure he would be perfectly willing for me to see them."

Dr. Mallory saw the sections from the first specimen later, but was, I believe, unable to make a more positive diagnosis.

May 22nd. No local recurrence. A gland, size of a bean, can be felt under the middle of the left side of the jaw. It is freely movable and not tender. On June 4th the gland had fully doubled in size, and it was decided to remove it. This was done, under ether anesthesia, on June 14. The pathologic report on the gland was as follows:

"June 25, 1915. The specimen consists of a moderately firm piece of tissue, measuring about 2 x 1.5 x 1 centimeters, and of a pinkish color. Microscopically it is composed almost entirely of spindle shaped fibrous cells, though there is an area of round cell at one border, and areolar tissue peripherally. Furthermore, while the spindle shaped cells do not vary a great deal in size or shape, their nuclei are often found in a state of mitosis. No epithelial cells whatever are apparent. Opinion: Although the tissue taken from the original tumor was determined as malignant, the metastasis gives a clearer conception of the cells; it is not a cancer, but a spindle (fibro) cell sarcoma. (Signed) DR. F. L. BURNETT."

Confirmed by Dr. Mallory.

No other glands could be felt at that time. The patient was next seen on July 7th. There was no local recurrence and no glands palpable. On July 30th there was no local recurrence, but a gland could be felt under the left sternomastoid muscle.

In view of the diagnosis of sarcoma, it was now determined to try the Coley treatment. This was carried out by or under the direction of Dr. Torr W. Harmer, and was continued for two months. The following account of the Coley treatment of this case and general comments are taken from a letter of Dr. Harmer's to me, dated March 2, 1916:

"When recurrence occurs in such cases and the toxins fail to influence this recurrence, further operation is usually unsuccessful. Regarding her (Mrs. X.'s) treatment under my care: I saw her August 10th, and gave her one-half minim of mixed toxins (Coley). Further treatments were as follows: August 12th, one minim; 16th, two minims; 18th, four minims; 20th, four minims. All injections were in the musculature of the upper back. All produced more or less local irritation; none caused marked reaction, although a few caused some elevation of temperature and chilliness. The treatments were then continued by Dr. Toppan of Newburyport, under my direction. He gave the injections every other day, then twice a week, reaching a maximum of fourteen minims. I then suggested that injections be given into the growth (meaning the gland under the left sternomastoid, J. P. C.), starting with a marked reduction in dose. The result with an injection of nine or ten minims was most distressing: marked fever, chill,

and vomiting, with temporary increase in size of tumor. A second injection into the tumor resulted in an equally distressing experience, and the toxin treatment was discontinued."

Dr. Harmer has had a large experience in this treatment of malignant tumors, and I shall give, at the end of this paper, a reference to a paper of his on this subject.

I saw the patient again on October 16th. Her general health appeared excellent, but the gland under the sternomastoid muscle was apparently unchanged, and there was a small recurrence on the cartilaginous septum, just below the perforation. This was a disagreeable surprise to me. I had been afraid of a recurrence on the outer wall of the nose, where I had cut rather close to the growth near its lower border, but not at the septal border, where a wide margin of healthy tissue had been taken. This recurrence was removed, under local anesthesia, on October 20th.

The report on this growth was as follows: "The specimen consists of a small piece of mucous membrane bearing a nodule about three millimeters in diameter, some cartilage and a sliver of bone. In a section of the soft tissue the cells are of an irregularly shaped spindle variety, with leucocytes and round cells scattered among them; but there also are some epithelial cells forming glands in the tissue and a covering of the tumor. The spindle cells vary in size and shape; some contain large, others small, nuclei, and now and then they are in a state of mitosis. The epithelial cells, on the other hand, are quite regular in size and shape. Opinion: The tumor is evidently a recurrence, as it is a spindle cell sarcoma."

The patient was next seen on November 13th. As the gland under the sternomastoid was apparently unchanged, it was decided, after consultation with Dr. George O. Clark and Dr. Harmer, to do a complete glandular dissection of the left side of the neck. This operation was performed on November 18th by Dr. George O. Clark. The dissection was done en bloc, and every vestige of glandular tissue removed. It is interesting to note that many glands were found involved which could not be palpated before the operation. The patient went home at the end of a week. There was a slight infection at the lower angle of the wound, where the dressing did not fully protect it. With the exception of this small complica-

tion, the wound healed solidly by first intention. There was a moderate left facial paralysis and some hoarseness (left recurrent paralysis?). I did not have an opportunity to examine the larynx at this time.

The subsequent history of the case is taken from Dr. Toppan's letters. The first signs of mental disturbance appeared on the night of December 5th, with rambling, incoherent talk. The next morning she had a convulsive attack with clonic convulsions, general in distribution, accompanied by rolling up of eyeballs, loss of consciousness for fifteen to twenty minutes, followed by pallor, a weak, thready pulse, and no remembrance of the attack. Subsequently the pulse, temperature and respiration were practically normal. The urine was normal, and there was no bowel trouble. For two weeks, following this attack, she was very restless, constantly moving hands or feet when awake, talking incoherently and irrationally, and sleeping poorly. She would start a sentence, but was, apparently, unable to finish it (motor aphasia). Her mind, however, seemed capable of understanding what was said to her during this time. She would answer rationally "Yes" or "No," but seemed unable to form words. The blood pressure (systolic) rose gradually from 150 to 180, going down again to 130 three days before death. Dysphagia increased slowly until even liquids were swallowed with difficulty, but later this symptom was not so marked. She lost weight and color. Reflexes were all apparently normal at first, and pupils reacted equally. She was drowsy at times and would sleep for the better part of two or three days, then wake and be very restless and irritable. About January 1st she practically ceased speaking and was more and more drowsy. It now became very difficult for her to walk, and by January 15th she was practically bed-ridden and had incontinence, worse of the bladder than rectum. There now appeared an exaggerated right knee jerk, with slight ankle clonus and some spasticity of the right arm, with twitching of right hand and arm and foot and leg. The right pupil was at this time slightly larger than the left, though both reacted somewhat sluggishly to light and distance. She became very comatose and hard to rouse, but would occasionally recognize her family. On January 17th the pulse became variable, ranging from 80 to 120 on different days. On January 22nd she sat up in bed and appeared very bright, but

could not utter an intelligent word. On February 1st the right knee jerk was absent, and there was beginning atrophy of the muscles of the right hand and arm, which she does not move, nor is there any twitching in it now. She ate practically nothing for the last ten days of life and died on February 4th, the immediate cause of death being, apparently, myocardial insufficiency.

In a letter dated February 6, 1916, Dr. Toppan says: "Am enclosing a rough sketch showing the general location of the masses taken from the brain of Mrs. X. Those shown at the base of the brain were directly under the pia mater, about equal in size, much firmer in consistency than the brain substance and easily shelled out from the brain with the finger. The mass shown in cross section in the left internal capsule was softer and looked almost necrotic in its center; it also was easily shelled out, was roughly rounded and about three-fourths of an inch in diameter. As a whole, the brain was rather 'wet.' So far as I could see there was nothing to indicate the course of metastasis. Vessels seemed normal everywhere." Dr. Toppan found no evidence of local recurrence. He sent the specimens to Dr. Burnett for examination, whose remarks and opinion on these growths follow:

"The specimen consists of three evenly rounded, soft masses in formalin; one measures about 2 x 2.5 x 2.5 centimeters, and was taken from the anterior portion of the temporal lobe on the left side; another from the internal capsule on the same side measures 2.5 x 2 x 2 centimeters, and the third from the cerebellum, 2 x 1.5 x 1 centimeters. In sections the tissue consists almost entirely of spindle shaped cells, in which a nucleus in a state of mitosis is occasionally apparent. Vessels are not especially numerous. Opinion: The tumors of the brain are sarcomata; and are evidently secondary growths from the tumor of the nose.

"(Signed) Dr. Francis L. Burnett."

Opinions confirmed by Drs. Graham and Mallory.

The very unusual and interesting features of this case are the metastases of the cervical glands and in the brain. While sarcoma of the nose often recurs again and again locally, metastases seem to be very rare. In the records of the Massachusetts General Hospital I find twenty cases diagnosed

sarcoma of the nose. Twelve of these cases were admitted to the wards, and eight were treated as out patients. In none of these is mention made of metastasis. Since 1896 fourteen cases of sarcoma of the nose have been reported in the Transactions of the American Laryngological Association, in none of which metastasis occurred. On looking over the literature I have found but eight of nasal or nasopharyngeal sarcoma in which metastases are said to have occurred.

In 1911, T. H. Farrell reported a case in which there was a small cervical gland, but this was not examined microscopically

Adair Dighton reports a case in a boy, eight years old, which he saw first in June, 1913, in which there was a hard polyp like mass protruding from the left nostril, slight exophthalmos of the left eye, and prominence of the left cheek. There were small tumors in the roof of the mouth at the junction of the anterior and middle third of the hard palate. The submaxillary, sublingual and deep cervical glands were enlarged and hard. The boy had had tonsils and adenoids removed two years before, and about one year later the cervical glands were found enlarged. The nasal condition was not discovered until one month before the patient was seen by Dr. Dighton. On removal, both the nasal tumor and the cervical glands were found to be round cell sarcoma. Dighton is of the opinion that the adenoids and tonsils, removed two years before, may have been sarcomatous, as the glands appeared a year after this operation and before the nasal growth gave evidence of its presence.

C. N. Slaney, in October, 1914, reported a case of multiple round cell sarcoma originating in the nares. The patient was a man, aged forty-three years, with a history of syphilis and gonorrhea. He had had nasal obstruction for three years or more. There was a large pedunculated mass filling the nasopharynx, but more on the right side. The tumor, very friable, was removed by snare under local anesthesia. A year later there were pains in both knees and shoulders, with edema of the feet, and both nostrils were obstructed. Two months later a small painless fluctuating tumor appeared in the frontal bone. Five months later the patient fell, owing to spontaneous fracture of the right femur and left tibia and fibula. Other tumors appeared under the scalp and in the fingers. Five months later the patient died from obstruction of the trachea by a

tumor. At the autopsy numerous erosions of the skull were found where the fluctuating tumors had been. There is no mention of a Wassermann test or of any syphilitic treatment, in spite of the syphilitic history. The microscopic report stated that the appearance was characteristic of round cell sarcoma.

It is well known that it is often impossible to differentiate a gumma from a round cell sarcoma by the microscope. The history of the case suggests that the multiple tumors were gummata rather than sarcomata. At all events, the diagnosis of sarcoma in this case is open to grave question.

J. A. Watson, in a study of one hundred and fifty cases, published in 1904, found metastases mentioned three times. In two of these cases the cervical glands alone were involved. These two cases have not been identified, as Watson does not mention them specifically.

Warthin's case, reported in 1899, the third case referred to by Watson, was a man, thirty-nine years old. A year and a half before seen by Warthin the left nostril became partly occluded, and a year later he had polypi removed from the nose which apparently were not considered malignant. For a month he had noticed numbness of the left side of the face. Diplopia, left sided ptosis, and dilatation of the left pupil soon followed. On admission there was weakness, nervousness, insomnia, and dull pains in the forehead. Nostrils were entirely occluded. The mind was clear. No operation was attempted. The condition became worse, and the patient died in coma six weeks after first seen. At autopsy a soft growth (size of a cherry) was found on the inner surface of the dura mater, near the longitudinal sinus. The pineal body and the hypophysis were replaced by tumor growth. The left third nerve and left Gasserian ganglion were completely surrounded by new growth. The soft tumor mass filling the nose was directly continuous with tumor masses filling the frontal sinus and sphenomaxillary fossa. The heart muscle was infiltrated by nodular tumors, and there was a large free tumor in the right ventricle. There were scattered small nodules in the lungs. All the abdominal organs showed metastases. The cervical lymph glands contained large tumor masses. The primary growth was a round cell sarcoma and the metastases showed a variety of pictures according to the age of the growth

and the tissues in which it was located. The primary growth was in the nasal fossa, without reasonable doubt, according to Warthin. "The richness of the growth in cellular elements, the scanty stroma, the numerous large thin-walled blood vessels, the smallness and shape of the cells, all contributed to the facility of the rapid dissemination of the tumor."

In 1914, A. Finzi gives an incomplete report entitled: "A case of tumor of the nasopharynx with metastases in the base of the brain and in the spinal cord." In this case, a man, aged twenty-two years, with severe headache and nerve symptoms, there was a malignant process starting in the nasopharynx. Besides the possible metastases in the brain and spinal cord, the cervical glands were enlarged. No operation was apparently done, and, consequently, there is no microscopic report. As the patient was still living at the time of the report, the diagnosis of metastasis in the brain and spinal cord was made solely from the symptoms. The tumor was assumed to be probably sarcoma.

Sendziac, in an exhaustive article, published in 1913, on malignant tumors of the nasal sinuses and nasopharynx, does not mention metastases.

Ferreri, in the same year, in a paper on the treatment and prognosis of malignant tumors of the nasal fossæ and nasopharynx, says nothing of metastasis.

In a paper on the Coley treatment of inoperable sarcoma, published in 1915, Dr. Torr Wagner Harmer gives the following report: "Case 25.—Multiple small round cell sarcomata. M. C., housewife, fifty-five years old. December 7, 1912. Six months ago began to have severe frontal headaches. Four months ago, small hard swelling on right side of nose near bridge, which had gradually and painlessly increased in size. Three months ago, mass discovered in front of right ear, which has gradually and painlessly grown. About the same time a mass appeared just above the right clavicle and another in right neck. Behind right sternomastoid muscle, several pea to bean-sized glands. In front and behind left sternomastoid muscle, several small glands, also palpable. . . . Excision of several glands for diagnosis. (Description of the toxin treatment is omitted. J. P. C.) Patient died with metastases in abdominal wall, chest and both breasts, in four months."

Omitting as doubtful or not proven, Farrell's, Dighton's,

Slaney's, two of the cases mentioned by Watson, and Finzi's cases, there remain only two cases besides my own in which the fact of metastases have been established, and only one with metastases in the brain. In my case the lymphatic channels through which the cervical glands became involved are quite evident. In describing the lymphatics of the face, Poirier and Charpy say: "The third group of lymphatics of the nose run with the facial vessels and terminate in the submaxillary glands. This cutaneous network is continuous with the lymphatics of the vestibule, and through them with the lymphatics of the mucous membrane. Anterior trunks of lymphatics run . . . . between the different cartilages, or even in front of them. They thus arrive at the celluloadipose subcutaneous tissue, where they unite into two trunks . . . . which terminate in the submaxillary glands."

Sobotta says that the submaxillary nodes empty into the superficial and deep cervical nodes. The natural course of the lymphatic stream thus accounts for the involvement of all the cervical glands in the anterior triangle of the left side.

The channel by which metastases reached the brain is not so clear. Quain's Anatomy says: "The lymphatics (of the nose) are abundant and large. They form a close plexus in the mucous membrane . . . . and a more open plexus of valved vessels nearer the bone. These are in communication with the lymphatic spaces which enclose the branches of the olfactory nerve, and these spaces again communicate with the subdural and subarachnoid spaces of the cranium so that the lymphatics of the nasal mucous membrane can be injected from the cranial cavity."

In the same connection, Poirier and Charpy say: "In the olfactory region of the mucous membrane of the nasal fossæ there are present, by the side of the lymphatic network, properly socalled, formations which are . . . . comparable to them. These are the meningeal sheaths of the first pair of nerves. Now, when these sheaths are injected through the subarachnoid space, it sometimes happens that the lymphatics of the nasal mucous membrane are filled at the same time. Axel Key and Retzius, who first noticed this fact, have never seen direct communications between the periolfactory sheaths and the lymphatics of the mucous membrane. . . . . Further study of the lymphatics of this region is important."

The fact that there were metastases in the meninges and brain in my case would seem to show that there may be a direct communication between the lymphatics of the nose and the interior of the skull. It would seem, from the rich lymphatic system of the nose, that metastasis in cases of sarcoma would be the rule instead of apparently the rare exception. What may be the reasons for this fact I am at a loss to surmise, nor do there seem to be any facts in the cases so far reported which throw any light on this question.

I desire to take this opportunity of thanking Dr. G. O. Clark for his surgical aid in this case, Dr. Toppan for his valuable contributions to the history, Dr. Harmer for his notes on the Coley treatment, and Drs. Burnett and Mallory for their careful pathologic reports.

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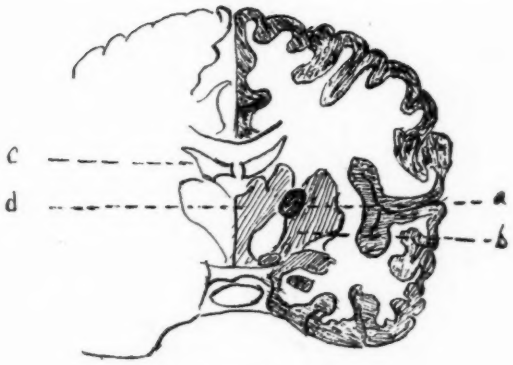

FIGURE 1.

Cross Section of Brain.

a.—Soft mass, roughly rounded, three-fourths inch in diameter, in the internal capsule.
b.—Lenticular nucleus.
c.—Lateral ventricle.
d.—Thalamus.

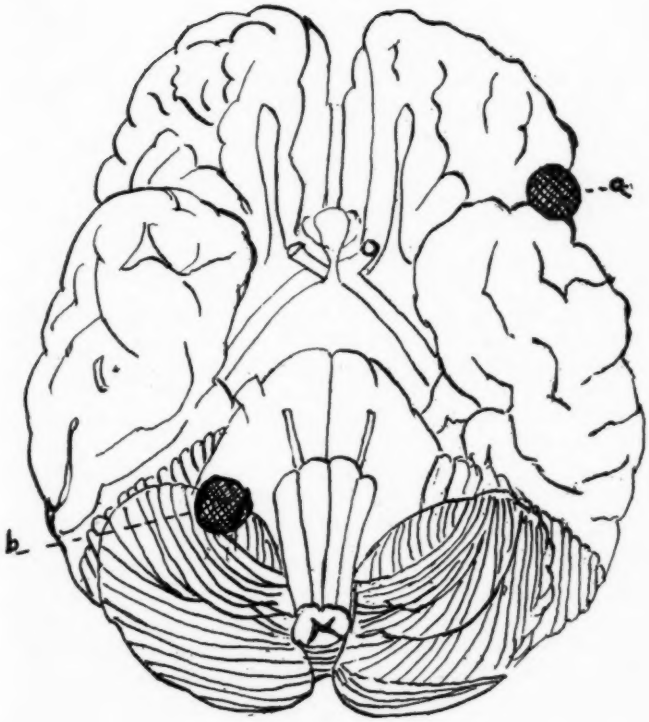

FIGURE 2.

a.—Round firm mass, one and one-half inches in diameter, at the anterior border of left temporal lobe at base of brain, just under the pia mater.
b.—Similar mass under the pia mater, just to right of olive, about where the vagus emerges.

XV.

DISSEMINATED MYELITIS AS COMPLICATION OF AN ACUTE MASTOID INFECTION.

By J. Ramsay Hunt, M. D.,

New York.

It is only too well known that cerebral complications occur very frequently after acute and chronic infections of the middle ear and mastoid region, in the form of inflammatory edema, meningitis, encephalitis and abscess. These are usually local manifestations, originating directly from the diseased focus and extending to adjacent areas of the brain substance and its coverings, through the blood and lymph channels, by direct contiguity from carious bone, or by traversing the sheaths of the facial and auditory nerves.

The central nervous system may also participate in a general pyemic process which sometimes occurs.

The form of infection to which I would call attention is one which, so far as could be determined by the clinical symptoms, was quite limited to the spinal cord—namely, an acute disseminated myelitis. This sequela of acute mastoiditis is of great rarity, if not unique in the literature of this subject, although it is well known that myelitis and encephalomyelitis

REPORT OF CASE.*

The patient is a woman, thirty-five years of age, a domestic servant by occupation. Her previous history is negative and without especial interest. She was in excellent health until April 1, 1915, when her right ear became painful following a "cold." The pain in the ear was very severe for two weeks, and was then followed by a purulent discharge from

*Presented at the Clinical Conference of the New York Neurological Institute, November 9, 1915.

the canal. On the appearance of the discharge the pain diminished in severity. After the ear had discharged for about three weeks the flow suddenly ceased, the pains in the ear and mastoid returned and became increasingly severe, and she applied to the New York Eye and Ear Infirmary for treatment. She was admitted to the service of Dr. Gorham Bacon on May 11, 1915, a diagnosis of acute mastoiditis was made and operative treatment recommended.

On May 13th Dr. T. L. Saunders performed a mastoid operation, which included exposure of the sinus and a considerable area of dura mater. Bacteriologic examination of purulent matter removed from the mastoid cells showed the presence of numerous colonies of streptococcus longus.*

On the fourth day following the operation she complained of indefinite pain in the back and lower extremities, followed by weakness and paresthesia of the trunk and legs. These symptoms were accompanied by fever and chilly sensations. Examination at this time showed some weakness and ataxia of the legs, and she was unable to stand or walk without assistance. Blood cultures made on May 18, 1915, were quite negative. The cellular constituents of the blood showed marked changes. Red cells, 2,200,000; white cells, 5,000; hemoglobin, 45 per cent.

Differential count as follows: Small mononuclears, 61 per cent; large mononuclears, 4 per cent; polynuclears, 34 per cent; eosinophiles, .5 per cent; mast cells, .5 per cent. The red corpuscles were irregular in size, with clear central areas. The urine was normal.

The pains in the lower extremities continued and soon extended to the neck and arms, especially on the right side. There was also a painful girdle sensation at the umbilical level, and a sense of constriction about the neck. Vesical symptoms and difficulty in defecation were also present. There was some headache low down in the occipital region, but no vertigo, delirium or other cerebral symptoms.

The mastoid wound healed without complication, but the spinal symptoms persisted and for a time increased in extent and severity.

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*Bacteriologic and other laboratory examinations were made under the direction of Dr. George S. Dixon.

Neurologic Examination.—On July 16, 1915, I first saw the patient, at the request of Dr. Bacon and Dr. Saunders. She was then almost paraplegic and unable to stand without assistance. There was marked static ataxia (Romberg symptom). She complained bitterly of pain in the neck and lumbar region, and there was a painful sense of constriction around the waist, with paresthesia of the hands and both lower limbs to the knees. The motor power of the arms was undisturbed, and there was no ataxia or tremor. The knee jerks and Achilles jerks were exaggerated, and there was a tendency to ankle and patellar clonus on both sides. The abdominal reflexes were elicitable, and the plantar reflexes were much diminished and of the flexor type.

There was marked disturbance of sensibility, both superficial and deep, of the lower extremities.

The pupils were equal and reacted to light and accommodation. Vision and the optic nerves were normal, and there was no nystagmus or paralysis of cranial nerves. Articulation was not affected.

At this time (June 16, 1915) a Wassermann test of the blood and spinal fluid was negative, and there was no increase of the globulin content, or of cellular elements. A blood count at the time showed some improvement over that taken on May 18th, but there was still severe anemia: Red cells, 2,160,000; white cells, 4,000; hemoglobin, 70 per cent.

Differential count: Small mononuclears, 53 per cent; large mononuclears, 10 per cent; polynuclears, 36 per cent. One nucleated red cell was found.

The diagnosis was made of a spinal cord affection, probably a myelitis of infectious or toxic origin. She remained in the infirmary until August, 1915, without material change in her condition, and was then transferred to the New York Neurological Institute, and was admitted to the Second Division (service of Dr. Frederick Peterson).

Her complaints on admission were as follows: Pain in neck and dorsal region, with broad girdle sensation about the chest and upper abdomen. Pain and painful paresthesia of the lower extremities and to a lesser degree of the arms, especially the right. Rather a constant area of pain in nape of neck extending to the right shoulder. Occasional incontinence of urine

and also of feces after laxatives. Inability to stand or walk. There is no complaint of headache, vertigo, diplopia, or visual disturbance. Her mind is perfectly clear.

Examination.—August 8, 1915. The patient is bedridden and incontinent. No decubitus. Pupils are equal and react to light and accommodation. No nystagmus.

The cranial nerves, including the optic discs, are negative.

The spinal column shows no deformity, but there is some tenderness on pressure at the level of the fourth D. and eighth D. spines, and there is very slight stiffness of the neck. Movements of the arms are free, and there is no ataxia or tremor. The grip of the right hand is somewhat weakened, and the right arm is the seat of pain and paresthesia, but without gross disturbance of sensation. The tendon reflexes of the upper extremities are present and not exaggerated.

Both lower extremities are spastic and paretic, the right more than the left. There is well marked patellar clonus and ankle clonus on both sides. The plantar reflexes are of the extensor type (Babinski). The abdominal reflexes are absent.

The postural sense of the toes is disturbed on both sides, and the superficial sensibility, touch, pain and temperature, is diminished or lost in irregular areas below the umbilical line, above which there is a broad zone of hyperesthesia.

On August 8, 1915, the Wassermann tests of the blood and cerebrospinal fluid were again negative, and there was no increase of globulin or of cells. The urine was negative.

The blood on November 6, 1915, showed considerable improvement over previous examinations, viz.: 4,782,000 red cells, with 70 per cent of hemoglobin. While under observation in the Neurological Institute there had been no fever, and the examination of the internal organs, heart, lungs and abdomen, showed no abnormalities.

COMMENT.

The patient remained in the institute three months, during which time there was a gradual abatement of the subjective symptoms with some return of power in the lower extremities. When discharged, November 15, 1915, there was still spastic paraplegia with paresthesias and a girdle sensation. The spinal cord symptoms had, however, shown a distinct improvement, no new symptom had developed, and the tendency was

to retrogression and not progression of the disease. There was, therefore, every reason to hope that the inflammatory process had been checked by the defensive forces of the body, and that eventually those neural structures which had suffered would gradually recover a considerable portion of their function. It is probable, however, that some weakness and spasticity will persist as a result of permanent injury to the pyramidal tracts.

As the spinal cord symptoms had followed so soon after the operative procedure, it was thought likely that the organisms (streptococci) or their toxins had entered some of the venous channels, thus reaching the general circulation. Just why the hematogenous infection should have become localized in the spinal cord is as much of a mystery in this case as in other of these obscure selective forms of inflammation. The possibility of a direct infection of the subdural space was also considered, with secondary invasion from the cerebrospinal fluid of the medulla spinalis. Such an invasion of the cerebrospinal fluid would be much more likely to cause meningitis, with the clinical and pathologic features of this disease, and while it is true that slight meningeal symptoms were noted, as spinal pain and some stiffness of the neck, these were not more acute than are commonly observed in myelitis, and the fluid obtained by lumbar puncture was free from any signs of inflammatory reaction.

The extreme degree of the infective and intoxication process is shown by the severe secondary anemia which was present. That this was merely a secondary anemia was evidenced by the marked improvement in the blood picture after the subsidence of the infection, and the funicular myelitis associated with pernicious and other grave anemias may, therefore, be excluded.

REMARKS.

This example of an acute disseminated myelitis is interesting and unusual because of the rare source of the infection—the middle ear and mastoid cells. The infectious myelitis, while not a common affection, is by no means infrequent, and may arise from a variety of causes. It is met with as an occasional complication after most of the acute infectious diseases of childhood, also affections like typhoid fever, osteomyelitis, erysipelas and during pregnancy and the puerperium. Not a

few cases have been associated with infections by the gonococcus and prostatic abscess; and it has been observed after such mild affections as panaritium, tonsillitis and angina.[1] Of more than passing interest are those cases which have seemed to develop after inflammatory and suppurative processes in the abdominal cavity—viz., appendicitis, metritis, parametritis and septic peritonitis—as it has been shown by Orr and Rows[2] that infections may travel along the lymphatic channels of the nerve sheaths directly to the spinal cord. Oppenheim[3] and Flatau[4] have each observed myelitis as a complication of suppurative processes in the antrum of Highmore. Bacteriologic examination sometimes shows the presence of such organisms as streptococci, staphylococci and pneumococci, both in the cerebrospinal fluid and postmortem in the cord substance. Typhoid and tubercle bacilli have also been found, but as a rule bacteriologic studies are negative, and it is assumed that the organisms are either present in very small numbers or that they have been destroyed and removed by the leucocytes. It is more than probable that the toxins of such organisms play an important, if not the chief rôle in the production of these forms of myelitis.

In many of the cases the symptoms and the pathologic lesions are by no means limited to the spinal cord, and the brain stem, cerebellum and cerebrum are involved as well—the encephalomyelitis. A rare and very interesting type is the acute optic neuromyelitis, in which retrobulbar neuritis and optic neuritis with amblyopia and amaurosis are early manifestations.[5]

A few of the cases may terminate in suppurative meningitis and abscess formation, but, as a general rule, the infection takes the form of a nonsuppurative myelitis or encephalomyelitis, as is not rare when certain strains of pyogenic organisms reach the deeper structures in small numbers—e. g., infective arthritis.

The prognosis in these cases depends upon the virulence of the infection and the extent and location of the lesions produced. Many of the cases terminate fatally in the acute stage; in others, serious cord disturbances are produced and death results in the chronic period from bed sores, secondary bladder and renal involvement. A not inconsiderable number of cases recover completely or with slight motor disability.

An interesting group of cases is that in which periods of re-

mission and intermission are observed, probably due to a recrudescence of bacterial activities. These constitute a borderline or transition type to the disseminated sclerosis.

In all those cases where it is possible to isolate an organism, treatment with autogenous vaccins should receive consideration.

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XVI.

BRONCHOSCOPIC AND ESOPHAGOSCOPIC POSTULATES.*

By Chevalier Jackson, M. D.,

Pittsburgh.

It has seemed to the writer that so many of the fundamentals of peroral endoscopy are, metaphorically speaking, in such a fluid state that the members of this society should formulate some sort of guidance for us all, to be modified to suit ourselves as applied to the particular case. The author is especially in doubt about the duration of bronchoscopy. Ingals, who is high authority, states that prolonged (over half an hour) bronchoscopies are dangerous. With this I heartily concur, but confess I, for one, do not know exactly where to draw the line. In the Pittsburgh clinic we are timing all our endoscopies, and hope eventually to accumulate valuable data. At present we try to work on the tentative basis of postulates three and four; but would be glad to have the views of others. Especially would we be glad if the safety limit of duration could be extended, because there are some difficult cases in which success seems so near that the utmost courage of one's convictions is required to desist and to postpone the bronchoscopy. Assuming that introduction of the bronchoscope does not require longer than one minute, all the time given is assumed to be devoted to bronchoscopic search and solution of mechanical problems of foreign body disimpaction and removal.

It is assumed that in recent, uncomplicated, untouched foreign body cases the mortality of bronchoscopy should not exceed two per cent. The simple passage of a bronchoscope in normal people as is said to be done in some clinics probably has no mortality.

These postulates are only tentative, to be discussed, rejected, accepted, excepted or modified, as experience may determine.

*Read before the American Laryngological, Rhinological and Otological Society, May 4, 1916.

Though they are phrased dogmatically, the author makes no claim to finality, and therefore begs to call particular attention to his intention to offer these formulations simply as postulates for discussion, not as rules to follow, except in case of such of the postulates as may receive your sanction; and even then it must be remembered that all good rules have exceptions. I hope also to have you add any postulates to cover omissions that doubtless have occurred.

1. Anesthesia should be left to the choice of the operator in the particular case. Some operators will prefer general, others local anesthesia. Others none at all in children. Every laryngologist should do diagnostic direct laryngoscopy in children without anesthesia, general or local. Very few endoscopists use any anesthesia, general or local, for esophagoscopy in adults. For ballooning the esophagus, as used by Mosher, general anesthesia is needful. Ether is preferable to chloroform. Cocain and morphin are dangerous in very young children. A few endoscopists use novocain and alypin.

2. An aseptic technic should be carried out as thoroughly as if the abdomen were to be entered by external incision. While absolute sterilization of the field is impossible, cleansing of the teeth and rinsing of the mouth with dilute alcohol should be attended to whenever practicable. In any event, we never need to introduce any organisms other than those already harbored by the patient.

3. A bronchoscopy ordinarily should not be continued longer than twenty minutes in a child under five years of age; nor longer than thirty minutes in one under twelve years. Unless for urgent reasons, a bronchoscopy should not be repeated at shorter intervals than five days in children under five years of age. Thirty minutes should be the time limit of an esophagoscopy in children under twelve years. Both bronchoscopy and esophagoscopy may be done in the newborn, if necessary, but the duration should not exceed fifteen minutes, except in case of bronchoscopy for the maintenance of respiration.

4. Four or more very careful twenty-minute bronchoscopies, at intervals of a few days, in an infant, are practically harmless; while one of an hour's duration in an infant may be fatal, no matter how carefully done. This does not apply to adults, in whom there is no special danger attached to prolonged bronchoscopies.

5. There are no absolute contraindications to bronchoscopy or esophagoscopy in foreign body cases. Many conditions may render inadvisable endoscopy for disease, and in foreign body cases may justify postponement of the endoscopic procedure. Aneurism, if positively diagnosticated, is a contraindication to bronchoscopy or esophagoscopy for disease, but not for foreign body.

6. In very dyspneic cases, if there is any lack of confidence as to the promptness of a bronchoscopy, a tracheotomy is advisable. Then the bronchoscope should be inserted through the mouth, not through the tracheotomic wound. The peroral route gives more freedom of movement, greater ease of searching the anterior branch bronchi, and less danger of infection. It is the route whose every movement is familiar from practice. No one can tell, in looking or working through a bronchoscope, with any of the modern systems of illumination, whether the tube is a long or a short one. In a dyspneic foreign body case the foreign body may be in the larynx, whence it would be most easily removed perorally, anyway.

7. The primary requisite for safe bronchoscopy is gentleness.

8. A foreign body should not be pulled upon until the bronchoscopist has a clear idea of the mechanical problem, nor until he is certain that the foreign body is free to be moved without inflicting trauma. To pull upon a foreign body without first making sure that it is disengaged and free to be moved is to court both failure and disaster. The problem is not simply to remove the foreign body, but to remove it without serious danger to the patient.

9. It is dangerous to pull on a tack, pin, needle, nail or other sharp pointed object until the point is in the tube mouth. Even then it is unwise to pull strongly. A child is better off alive with a tack in its lung or esophagus than dead or dying with it out. In most cases of foreign body in the bronchi the patient will live many months if no trauma is caused by attempts at removal. Hence, hasty, desperate, ill studied, violent attempts at removal are unjustifiable.

10. Full-curved hooks are to be used in the bronchi with greatest caution, if used at all, lest they catch inextricably in branch bronchi.

11. Laceration or perforation of the thoracic esophagus is almost invariably fatal.

12. The watchword of the bronchoscopist should be, "If I can do no good, I will at least do no harm."

XVII.

CARTILAGE AND BONE IN THE TONSILS.

By W. E. Grove, M. D.,

Milwaukee.

Although our knowledge of the physiology of the tonsil is but fragmentary and incomplete, the pathology of this organ is very well known. We know that the tonsils are very often predisposed to infection, both acute and chronic, and comparatively seldom to tumor formation. Whether we shall class the formation of cartilage and bone in the tonsillar tissue as of neoplastic origin, or whether we shall regard such changes as due to the results of chronic infection, will depend largely upon our view as to the origin of such pathologic changes.

We know that the tonsil consists, in general, of collections of lymphoid tissue, arranged in follicles, grouped around crypts and separated from each other by trabeculæ of connective tissue derived from the fibrous capsule which surrounds it. This structure is covered on the exposed mouth side by a stratified pavement epithelium. These tissues, together with the blood and lymph vessels, form the histologic elements and tissues of which the tonsil is normally composed. When any other tissue, such as bone or cartilage, is found in the substance of the tonsil, it must be regarded as pathologic and must be explained as being either metaplastic or neoplastic in origin, or as being a misplaced embryologic rest.

As early as 1898 Walsham called attention to occurrence of cartilaginous and bony nodules in the tonsil. While making observations on the occurrence of the tubercle in the tonsil he came across two tonsils in which he found both bone and cartilage.

The first was from a man aged fifty years, dead of pulmonary tuberculosis. Both tonsils had undergone considerable atrophy. Scattered throughout the organ, at the base of the

cypts, were numerous small masses of bone in the form of trabeculæ, rings or solid nodules. They did not encroach upon the adenoid tissue.

The second case was a man aged twenty years, dead of chronic pulmonary tuberculosis. In this case large masses of cartilage were scattered through the adenoid tissue. In some areas it had been converted into bone. The matrix was in some instances of hyalin, in others of fibrocartilage, variety. The nodules were surrounded by a dense fibrous tissue, forming a kind of perichondrium. Some of the nodules penetrated the adenoid tissue, in one instance almost reaching the epithelium.

These two cases were found in a consecutive examination of thirty-four postmortems.

Walsham assumes that these cartilaginous masses were embryonic rests from the fetal cartilage of the second branchial arch. Professor Kanthack, to whom he showed his specimens, dissented absolutely from this theory, and believed that the cartilage and bone formation were due to a metaplasia of fibrous tissue.

Wingrave, publishing in the same volume of the *Lancet*, first found such cartilaginous nodules in the microscopic examination of diseased tonsils, mostly suffering from a chronic hypertrophy, removed from patients under twenty years of age. This cartilage was both hyalin and white fibrous in type, imbedded in a dense connective tissue capsule, and never occurring in the lymph nodules or follicles. In one instance calcification was noted, but this he did not regard as truly osseous tissue.

In ten tonsils removed from cadavers, Töpfer found cartilage in all three times. He also found in a tonsil removed from a case of benign mycosis tonsillaris a number of areas of cartilage, always surrounded by connective tissue, and never in the midst of adenoid tissue. In one tonsil removed from an old man he found an area which he believed showed a direct gradual transition from the fibrillar connective tissue. The center of the cartilaginous area was hyalin. Around this was a transition zone, which he designates as "Netzknorpel," in which zone he believes the connective tissue was gradually being transformed into cartilage. He regards the hyalin car-

tilage as the finished product, and the fibrocartilage as a "vorstage."

Nösske found cartilage and bone in six cases; five times in both tonsils, and once in one tonsil only. The ages of these cases ranged from thirty-seven to seventy-six. These were found in various parts of the tonsil, but mostly in the deeper layers of the connective tissue septa. Numerous transitions from connective tissue to cartilage and bone could be made out. Nowhere did the cartilage or bone show any tendency to get out of the connective tissue. In all the cases atrophic and regressive changes were noted in the tonsils, mostly in the connective tissue septa.

Among others who have reported the finding of cartilage and bone in the tonsillar tissues are Schweitzer, E. Zuckerkandl, Imhofer, Munzer, and Reitman.

In order to determine whether we were dealing with an embryologic anlage of cartilage, Reitman examined in serial sections three human embryos of fifty-one, sixty and seventy-two millimeters in length. Furthermore, he examined serially fifty tonsils from thirty-seven individuals of all ages. In all, seventeen tonsils, or thirty-four per cent, gave a positive finding.

In none of the three embryos could he find anything definite, though in the embryo of sixty millimeters he found a piece of cartilage at the end of the styloid process, and in the position of the still undeveloped tonsil.

He found cartilage in the tonsils of four newborn infants, in one child of one-half year, in one of one and one-half years, and the other cases varied from six to thirty-three years. In most of these cases he had to deal with hyalin cartilage, less frequently with the embryologic type of cartilage, rich in cells. All of the cartilaginous foci were distinctly separate from any connection with the skeletal tissues.

My attention was first called to the subject by finding areas of cartilage in sections of the tonsils removed from a case of benign mycosis of the tonsils. The age of this patient was sixteen years. The tonsils were very small and atrophic, and the crypts were filled with the typical masses of mycelium of the fungus growth. The lymphoid tissue was greatly reduced in amount. The cartilaginous areas were found near the posterior capsule and in the connective tissue of the framework,

having no connection with the lymphoid tissue. (Figure 1.) The cartilage was definitely hyalin in character at its center, and near its edges seemed gradually to pass over into the connective tissue of the tonsil by means of transitional fibrocartilage. (Figure 2.) No definite capsule could be made out, and at the edges of the cartilage, as in Figure 2, which is an enlargement of part of Figure 1, the direct transition from the connective tissue framework of the tonsil to the hyalin cartilage can be made out.

The second case, from my own practice, occurred about six months ago, in a young woman of twenty-three years, suffering from a chronic tonsillitis. The tonsils were slightly enlarged and the crypts were full of foul-smelling, cheesy plugs. The enucleation was very difficult, a snare wire being broken in removing the right tonsil. In passing the finger over the capsule of the tonsil the hard nodules could be plainly made out macroscopically. Sections of these tonsils show the numerous areas of cartilage. These are all of the hyalin variety. They are all situated in the connective tissue of the organ. They do not seem to have a very definite capsule, but one can make out the gradual transition into the fibrous connective tissue of the trabecular framework.

In running over the sections of the tonsils which we have removed at the Milwaukee County Hospital during the last two years, I found one other specimen. Undoubtedly, in working over this material we would have found many other instances had we made serial sections and examined every fifth or tenth section, as was done by Reitman.

CONCLUSIONS.

It would seem from these isolated cases, and from a study of the literature on the subject, that the presence of cartilage, and even bone, in the tonsillar tissue is not of uncommon occurrence. It would be found much more commonly and frequently were all tonsils removed, sectioned and systematically studied.

The cartilage found is true cartilage, and is of the hyalin and fibrocartilage varieties. It is uniformly found near the capsule or in the connective tissue framework of the organ. It never invades the lymphoid tissues. It has been found in all ages, but more usually in the tonsils of adults. It has most

commonly been discovered in tonsils which were either undergoing regressive changes or which were the subject of chronic inflammatory changes; less frequently in the healthy normal tonsils of children.

Whether the bone tissue occasionally found in tonsils is true bone, or whether it is a calcification of preexisting cartilaginous tissue, is a question I cannot enter into with the material at my disposal. I am, however, inclined to think that true osseous tissue has never been found in the tonsil.

The theories offered to explain the presence of the cartilage are three: First, that it takes its origin from the fetal cartilage of the second branchial arch; second, that it is a remnant of misplaced embryologic rests; and third, that it is the result of a metaplasia of connective tissue occurring in tonsils which are the foci of chronic inflammatory processes, or which are in the process of atrophy.

If the cartilage occurring in these tonsils takes its origin from the fetal cartilage of the second branchial arch, or if it is a rest of embryologic cartilage, misplaced, we would naturally always expect to find only one variety of cartilage. As a matter of fact, we find two varieties of cartilage: fibrocartilage and the hyalin variety.

If the cartilage is a remnant or rest of misplaced embryologic cartilage, why should we uniformly find the foci only in certain regions of the tonsillar structure, and always bearing a definite relation to the connective tissue framework, as Töpfer points out?

Moreover, if the theory of misplaced embryonal cartilage be true, then we must expect to find the cartilaginous areas as frequently in the tonsils of children as in those of adults. This is not the case, for Nösske was never able to find these changes in children, and Reitman, although he did find the changes in children, was not able to find them as frequently in infants and embryos as in older children and adults.

It seems much more logical to me to explain these changes as a metaplasia or a metamorphosis of connective tissue, due to inflammatory, hypertrophic or atrophic regressive changes in the tonsil. The reasons for this are:

First, that the cartilage always occurs in definite relation to the connective tissue. This was true in my cases and in all the reports in the literature.

Second, these areas of cartilage never exhibit any true perichondrium about them, and can usually be shown to merge by gradual transition into the connective tissue surrounding them. This can be made out in my first case in Figure 2, which is an enlargement of a portion of Figure 1, where the hyalin cartilage first passes over gradually by means of fibrocartilage, and then a dense connective tissue rich in cells, into the normal connective tissue of the tonsil. In fact, we must consider the hyalin cartilage as the finished product in these cases, and the fibrocartilage as the "vor-stage" in its formation.

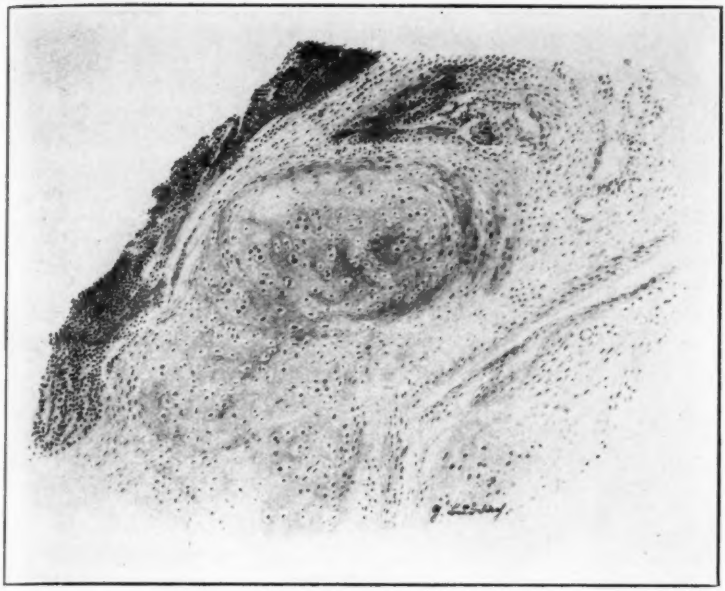

FIGURE 1.

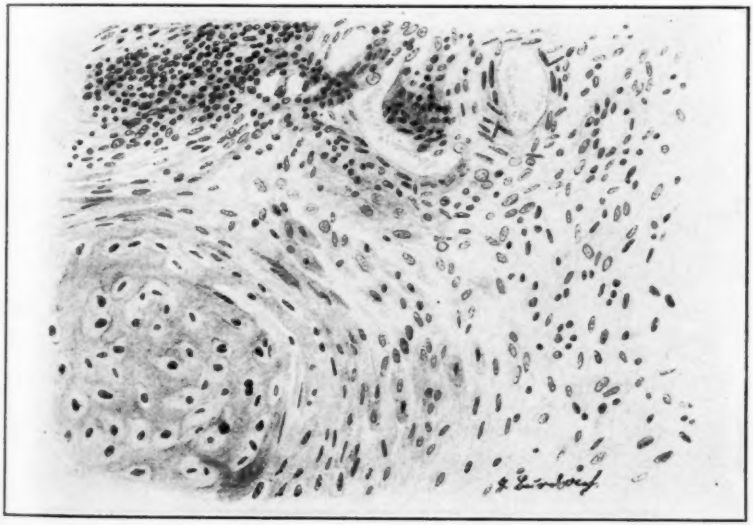

FIGURE 2.

XVIII.

AIR AND RHINITIS.

By Walter J. Wurtz, M. D.,

Buffalo.

In considering rhinitis we often refer to it as "cold in the head." In this reference we cannot confine ourselves to one disease. The term "cold in the head" is vague, but not entirely meaningless. The term was coined many years ago and is here to stay. If we would only think of the meaning of the words "catching cold," we probably would not use them as often as we do. How often have all of us here told our patients that they have "caught cold," when their wound suppurated, or "caught cold" where there was a relapse in some febrile condition, or following an obstetric case. However, it only means one explanation—that is, to satisfy the laity.

In the etiology of "cold in the head," I believe hygiene to be one of the first considerations. Hygiene of the body as well as hygiene of the home, schools and other public buildings must come under our observation. Under hygiene of the body we must look well to our eliminative organs and digestion, keeping the skin active and bowels regular, thereby lessening the chances of an autointoxication. The body should be well protected but not overburdened with clothes, so that radiation is assured. Some authorities advocate the same weight underwear all the year round, and increase the top clothing as to weather conditions. The hygiene of the home and public buildings should be looked after, and is of equal importance. The air should be changed often and proper humidity maintained. The average humidity of a room should be from forty per cent to sixty per cent, but in our climate it cannot be over twenty per cent, due to the extreme cold which lowers the dew point. The humidifying of air and the removal of dust from factories has improved some factories so that the attendance of their employes has been close to one hundred

per cent, where formerly it would be from sixty and seventy-five per cent because of the "colds" they "caught" during inclement weather. The subject of ventilation, with its kindred branches, humidity and dust elimination, has become a study which is employing the serious consideration of scientists the world over.

Volumes have been written, large sums expended in experimentation by individuals and corporations, municipalities enact and enforce ordinances for the betterment of conditions, arichtects and engineers strive to plan and construct heating and ventilating plants to give the best possible results, and engineering and medical societies are giving a full share of their time to this work. All these have made great strides in the right direction, but still there remains much to be done.

The ventilation and sanitation of large buildings, such as office buildings, hospitals, schools, churches, theatres and factories, must be considered as to the purity of air and proper humidity. The question naturally arises as to "what is fresh air." From a ventilating standpoint, fresh air is air free from dust and bacteria and a minimum amount of carbon dioxid (CO_2).

Ordinary fresh air, or outdoor air, contains on an average four parts of CO_2 in ten thousand.

Mr. Chas. S. Churchill reports as low as 1.76 to ten thousand in Paris, and 1.75 in London. These are unusually low, and may be due to the method of determination, as the records of the Montsouris Observatory at Paris gave as an average of observations covering thirteen years, 3.14.

Dr. G. A. Soper reports an average content for two thousand observations of New York City air of 3.61 and a maximum of 5.61 and a minimum of 2.69.

CO_2 is harmless, even in the most poorly ventilated rooms. The feeling of depression is usually due to high temperature and high relative humidity.

Dr. E. Vernon Hill, in the report of the ventilating division of the Chicago Health Department, says: "Repeated investigations by physiologic chemists have failed to find any poisonous or harmful ingredients in respired air, but the fact should be emphasized that while such substances have been found, it would be unscientific and illogical to assume that they do not

exist. We must, however, discard the belief that CO_2 can be used in any way as a measure of such impurities."

Also CO_2 is a very accurate measure of the amount of fresh air supplied, and by reason of the fact that fresh air bears a direct relation to the bacteria in the air, it is in this way a reliable index to the dust and bacteria.

A room is considered to be fairly well ventilated when the CO_2 is not over six or eight parts in ten thousand.

The amount of fresh air to be supplied per person has been placed at different figures.

Ohio's new building code requires a ten minute air change for churches, lodges, clubs, hospitals or hotels. All other assembly halls and theater auditoriums, not less than twelve hundred cubic feet of air per hour for each person.

Dr. Hill conducted a series of experiments in examining the air collected in public places, also took cultures by exposing Petrie dishes in these places, and examined as to the variety of germs and CO_2. All these showed a great deal of CO_2 and dust in the air.

Further experiments were made where a room had absolutely pure air and an employe was set to work therein. Gradually CO_2 gas was forced in the room, replacing the oxygen. The CO_2 was raised to the point where it was thought no person could exist very long. The man, however, stayed there several hours, and the only thing he noticed was a fullness in the head. He was again placed in the room and dust was forced in; he then developed an acute coryza and could not stay as long as before. This is only to prove that clean bad air has little effect, but the colds we receive are from infected as well as bad air.

Benjamin Franklin in his essay says that "colds result neither from wet nor cold, but from other causes entirely independent of this malady."

Robt. Peary in his diary states that none of his party developed "a cold" in their dash to the North Pole, but they all were attacked a few days after they returned to the settlement. This detail has been gone into only to show the importance of hygiene in "colds in the head."

Dr. Chas. D. Graham-Rodgers, of the New York State Labor Department, recommends for some factories ventilation

as high as from three thousand to five thousand cubic feet of air per hour per person.

There are cases in which the amount of air per hour per person is varied to suit the circumstances, a few of which follow:

Hospitals (ordinary)	2100 to 2400
Hospitals (contagious)	4800
Workshops	1500
Prisons	1800
Theaters	1200 to 1800
Meeting halls	1200
Schools (for children)	1200
Schools (for adults)	2400

Fresh air may be supplied in sufficient quantities, but if too dry, disagreeable sensations are experienced. Therefore, it must contain a certain amount of moisture. The amount of moisture which the air will hold depends on the temperature. For instance, at 70° it will hold eight grains per cubic foot, while at 32° it will hold two grains, and at 0° only one-half grain. Since air normally has a humidity of from forty to fifty per cent, air at 70° should contain from three and one-half to five and one-half grains, and at 32° about one and one-fourth grains. But when taking outside air for ventilating in the winter at 32° and heating it to 70°, it would have a humidity of only fifteen and one-half per cent; or if taken in at zero, it would have only from three to five per cent, a condition dryer than that of any desert.

Dr. Henry Mitchell Smith, in an article entitled "Indoor Humidity," says: "The overheating of our houses has been accepted as a prominent cause for catarrh, but I am confident that the low relative humidity and consequently the large saturation deficit of the aqueous vapor in the atmosphere of our rooms is much more important." Also, "It is easier to 'take cold' in a room at 72° with a relative humidity of thirty per cent, than in a room of 65° with a relative humidity of sixty per cent."

Dr. W. A. Evens proposes the following for school ventilating: "First, reduce the temperature of rooms to 68°. In such a temperature the exhaled air, being hot and moist, will rise out of the breathing zone and be replaced by purer air.

Second, raise the relative humidity to sixty per cent or seventy per cent. If the relative humidity is raised to sixty per cent, the pupils will be comfortable with a temperature of 68°. Maintaining a moderate humidity tends to keep the dust inactive and renders persons less subject to draughts. Third, the amount of dust in the air depends on climatic conditions and local influences. There is more dust in a manufacturing district than in a residential section, and less in the country. There is also more dust in the summer than in the winter."

Bruce L. Cushing, to whom I am indebted for the engineering data, recently made some tests on indoor and outdoor air, with the following results:

"In a tight room with a temperature of 70° and a relative humidity of fifty-five per cent (unwashed air, in motion) there was one milligram of dust in every thirty cubic feet of air.

"With the ground covered with snow, an average temperature of 40° and a humidity between forty per cent and fifty-five per cent, there was only one milligram of dust to two hundred cubic feet of air. The latter seemed to be mostly soot or factory smoke."

These were long-time tests, taken with an apparatus which was constructed under Mr. Cushing's supervision, which is, to his knowledge, the only accurate long-time testing instrument ever used.

Dr. Hill places the maximum amount of dust for good ventilation at 4.2 particles per cubic foot, and the number of bacteria colonies on a five minute plate not over twenty.

To insure a supply of clean pure air for ventilation, an air washer is necessary. (An air washer is a metal case containing, in the order named, a distributing plate and one or two banks of spray nozzles and eliminators. Under this case is located a water tank, from which the spray water is pumped to the nozzles and to which it returns.)

There are three primary requirements in the design of an effective air washer and humidifier:

First.—The Spraying System: (a) Must be uniformly distributed to treat all parts of the air currents alike. (b) It must freely atomize the water and thoroughly mix with the air, in order to wet and make heavy the smaller or minute particles of dust and dirt carried by the air. Large drops of spray

or sheets of water will not wet these smaller particles. (c) The nozzles should discharge in the direction of air flow, so a minimum resistance is offered to the air current. (d) The spray nozzles should have large, unobstructed orifices, so they will not clog. (e) Proper and adequate filtering must be provided, to prevent dirt and other obstructions from entering the spray piping and nozzles.

Second.—Washing Surface: The removing of dirt, dust, soot and bacteria from the air is almost proportional to which it is exposed. Some of the heavier solid matter is precipitated to the tank by the spray, but the smaller particles, even after wetting, are carried in suspension by the air currents. In order to effectively remove this very fine material, the air must be broken into thin layers or strata and passed over a surface kept flooded with running water. This surface should be arranged to provide changes in the direction of air flow in order to throw the dust particles against it and wash them to the tank.

The Carrier air washing and humidifying apparatus is being installed in many of our modern buildings and schools. A public school so equipped was situated next to a garage. The odor of gasoline was constantly in some of the rooms. After the installation of an air washing and humidifying apparatus there was deposited in the receiving pan a layer of mud, a layer of cylinder oil, and a layer of gasoline, all collected from the air of this school. These washers remove ninety-eight per cent of the dust, dirt and bacteria from the air. Five pails of dirt were washed from the air used for ventilation of Public School No. 6, Brooklyn, New York, in one week. No doubt equally as much more was washed out when the tank was drained. One hundred and twenty-five pounds of dirt, carrying disease, some of which otherwise would have been inhaled by the pupils and the balance have settled on the furniture or been kept in circulation, was certainly worth eliminating.

The varieties of "cold in the head" depends entirely on the diagnosis. The varieties which we meet are: acute, chronic, caseous (inflammatory or suppurative), diphtheritic, and syphilitic. Of the chronic form we have the hypertrophic, atrophic, and polypoid degeneration, acute and chronic sinuitis, foreign

body in the nose, etc. Any of the above mentioned conditions produces an effect on olfaction, phonation, and respiration.

The main physiologic function of the turbinate bodies is to warm and moisten as well as to filter the air. The mucosa of the nose has tremendous power for radiation and moistening the air. The estimated amount of secretion in twenty-four hours is supposed to be about one pint. Cold air entering the nose is warmed at body temperature by the time it reaches the larynx. The physiologic function of the accessory sinuses of the nose is probably to promote resonance and also to dissipate shock in case of injury to the cranium. The specific duty the ethmoid cells perform is the nonconducting of heat or cold. The importance of this is recognized when we can see how the delicate structures about the orbit are protected.

In an acute rhinitis the patient's first complaint is dryness of the nose, slight temperature, and probably some headache. This is soon followed by a serous discharge. The mucosa in the beginning is engorged, swollen and dry. Later, following the transudation of serum from the blood vessels, the mucosa is still swollen but bathed in moisture. The discharge soon becomes viscid and involution soon follows.

In chronic rhinitis the patient complains of heavy sensation in the forehead, listlessness, and mucopurulent discharge. The mucosa is swollen and may have the appearance of tumefaction. It is pale, and the discharge adheres to the mucous membrane. On cocainizing, the membrane shrinks tight about the turbinates. This condition is also known as intumescent hypertrophy.

In true hypertrophy the membrane appears pale, discharge is viscid, and on cocainizing the membrane does not shrink about the turbinate bodies.

In all cases of rhinitis it is difficult to say where rhinitis ends and sinuitis begins.

With all the foregoing conditions a sinuitis may have been superimposed. We rarely see a true simple rhinitis without an accompanying sinuitis. In all inflammatory conditions of the nose, consideration must be given to deformities, foreign bodies, and tumors. By tumors we refer to the various polypoid degenerations, fibroid tumors and other neoplasms. Only recently I had occasion to see a patient with a fibroid tumor

that filled the entire nasopharynx and extended down close to the epiglottis. The uvula was pushed over to the right side of the mouth, lying against the jaw. This condition causes a degenerating process to go on in the nose due to the mechanic force—pressure.

In diseases of the accessory sinuses of the nose that give rise to various forms of rhinitis, we must consider the maxillary antrum, ethmoid cells, frontal sinuses, and sphenoid cells. In diseases of the maxillary antrum, proper drainage is the thing to accomplish. If polypoid degeneration has taken place, the antrum must be opened radically and all polypoid degenerating tissue removed. In ethmoid disease always be sure of the diagnosis, for the physiology of the cells must be kept in mind. As nonconductors of heat or cold, their protection of the orbit is necessary, therefore, complete exenteration should be done only when infection is beyond medical treatment.

Early in ethmoid disease stimulating treatment will produce marvelous results. In complete destruction of the ethmoid cells the middle turbinate is always sacrificed. The sense of smell is sometimes impaired by too radical an operation. Often, however, the sense of smell has been destroyed by preceding rhinitis.

In frontal and sphenoidal sinus disease, drainage again is the important factor. In operating on the sinuses one must always be prepared for various malformations, as no two sinuses are alike. In all long-standing discharges of the nose, where the discharge has been profuse, we often see hypertrophy of the nose and upper lip, with excoriation of the skin of those parts.

There is also a condition described by Vaughn in which the mucous membrane assumes a pale appearance and very much swollen. This often follows certain attacks of asthma and hay fever. He attributes this to protein poison in the circulation, producing a toxin affecting these parts. If we can get more information as to the character of these toxins we may soon have a remedy for hay fever and certain cases of asthma.

In conclusion, I wish to emphasize the importance of mak-

ing a differential diagnosis when we see a case of "cold in the head," and also the importance of hygiene in its relation to the onset and cure of this malady.

I am indebted to Mr. Bruce L. Cushing, C. E., for the accurate data pertaining to the engineering portion of my paper. He is associated with Mr. Carrier, the inventor of the air washer and humidifying apparatus.

XIX.

INTUBATION.*

By U. S. Bird, M. D.,

Tampa.

Intubation, in the treatment of infantile laryngeal diphtheria, stands alone as the most dramatically effective procedure in medicine. The change wrought by the tube is amazing, especially to the novice. It was formerly unique in its usefulness, but that has been abridged by the advent of a method without a superior in medical practice. A large factor in the usefulness of antitoxin is its freedom from the spectacular details incident to intubation, an operation requiring some degree of trained skill, while the serum, if necessary, may be given by a layman. At first thought it is almost regretted that so successful a procedure should have been so entirely eclipsed in its zenith and relegated to the department of rare operations; but a second thought accepts the situation with a profound appreciation of the beneficent efficiency of the serum. While we may expect improved methods of treatment and education to progressively limit intubation, it will not disappear entirely. There will always be a small but certain number of severe or neglected cases requiring instrumentation. Following is a brief record of my cases:

Case 1.—In November, 1907, I was called by Dr. O. about noon to see a dyspneic boy, about two years old, with diphtheria. The case was not urgent, and we kept it under observation till midnight, when a tube was placed on the third attempt, with immediate relief. Next day the tube was expelled and was replaced by a larger size, on the first attempt, an hour later. This was coughed up three days later, without unpleasant result, recovery proceeding uninterruptedly.

Case 2.—In March, 1908, I was called by Dr. B. to see a

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

case with the usual history. The boy was four years old and in poor general condition. A tube was placed on the third attempt, the case proceeding without incident. This case terminated unfortunately. The tube was removed at the second attempt, on the fourth day, the larynx being and continuing in good condition; but death occurred within a week from gastro-enteritis.

Case 3.—Seen with Dr. B., May, 1910. This case, a boy of two years, was uneventful, excepting the laryngeal emergency. The tube was placed on the third attempt, and removed on the first attempt on the fifth day, recovery being without incident.

Case 4.—This was a serious case, seen December, 1912, with Dr. R. The boy, four years old, was exhausted and languid, respirations shallow and difficult. Three attempts were necessary. When I was satisfied that the tube was in place, respiration had ceased, and he was cyanosed and limp. He was hastily placed on a table with head over the edge, and artificial respiration used for a minute before it was responded to. Recovery proceeded rapidly, and in twenty minutes he was sitting up and taking milk. This was the only case in which I left the cord in place, attached to the cheek by adhesive plaster. The tube was removed five days later, to be replaced two hours later, at the third attempt, again leaving the cord. Five days later it was removed permanently.

Case 5.—This was the most troublesome of my cases. I was called by Dr. B. to see a girl of two years, December 20, 1912. It was a neglected case of diphtheria with labored respiration. The tube was placed on the third attempt, with rather delayed relief. On the sixth day the tube was removed on the second attempt, being replaced two hours later, on the first attempt. On this occasion the doctor was absent, and I placed the tube with the assistance of members of the family, with little trouble. On this and subsequent instrumentations of this case, one attempt sufficed. Six days later it was removed on the first attempt. In the light of the previous experience I anticipated about the same interval in case replacement should be necessary. I hurried back an hour and a half later, in response to repeated calls by the family and the doctor, finding the baby in much distress, which was relieved by a hurried intubation. Three days later the tube was expelled and a

larger size used, which was retained two days, and replaced by a still larger size. This was expelled after four days, but the condition remained satisfactory thereafter. On this case I did five intubations and three extractions. I see the patient occasionally. The voice is rough, but has improved lately.

Case 6.—The treatment in this case was brief. In March, 1914, I was called by Dr. F. in the forenoon to see a diphtheritic, dyspneic boy of three years. A tube was placed on the second attempt, to be expelled an hour later, and replaced by a larger tube on the first attempt, which was expelled on the following day. The condition being satisfactory, the case was left with the attending physician.

Case 7.—This was a case according to the rule. In April, 1914, I was called by Dr. S. to see a boy of three years, with the usual history and condition. A tube was placed at the third attempt, left in five days, and removed at the first attempt without subsequent incident.

Case 8.—This was another case giving little trouble. I was called by Dr. I. on a morning in last June, to see a boy of two years. A tube was placed on the first attempt, to be expelled an hour later, and be replaced by a larger size, on first attempt, which was expelled three days later without further incident.

My experience in this small series of cases has developed nothing out of the ordinary. There were no deaths from the condition treated. The only one in the series was Case 2, from a trouble not related to diphtheria. The eight cases represent sixteen intubations and six extractions. In six cases three attempts were necessary for the initial intubation; in one, two attempts; and in one, one attempt. In the four cases in which one additional intubation was done, one required three attempts; the other three, one each. In the case requiring four additional intubations, one attempt each sufficed. I had little trouble with my extractions, one or two attempts sufficing for each.

The technic is well known. The guides are the epiglottis and the arytenoids. Sometimes the latter only can be distinguished; the epiglottis imparting a mushy sensation to the finger, indicative of nothing in particular. The Feroud instrument is the only one satisfactory to me. I have bent the obturator and removed the tube, after having placed it, in removing the O'Dwyer intubator. Although I have been prepared

for tracheotomy on occasions of intubation, my cases have developed no condition requiring it, and I anticipate no such trouble. If membrane should be pushed down and stop the tube, its prompt removal by the cord may be expected to relieve the emergency. Contrary to cautions lately appearing regarding the use of too large tubes, my trouble has been to get a tube large enough to stay in. In one case I twice increased the size of the tube, and the last was finally coughed up. I would not consider replacing the same tube, unless in a hospital where help was always at hand. Even then it would be a waste of time. All of my cases were at their homes. I have had little trouble with nourishment. Nursing has always been possible. I have given nothing but liquids, and they were usually taken from a spoon, in any posture. Careful preparation is desirable, but these cases are emergencies, and it is surprising how little preparation and assistance suffice. Sterile instruments are necessary, and a gown and gloves, also sterile, are a comfort, but on occasion formalities may be dispensed with without risk to the patient.

Two reliable persons, one to sit and hold the child, the other to hold the gag and head, are usfficient. On one occasion my preparation consisted in removing my coat, rolling up my sleeves, rolling the child in a sheet, placing it in the lap of some one in a straight chair, adjusting the gag and getting some one else to hold it and the head. Within three minutes after I entered the house the tube was in place, and I indulged in the luxury of a long breath; my removal of the tube two hours before was the cause of the emergency.

Since writing the above two other cases have come under my care.

Case 9.—On November 8, 1914, I was called by Dr. B. to see G., a girl, eight years old, the oldest of my cases. She was in distress, having been kept awake some thirty-six hours by respiratory efforts. A tube was placed on the third attempt, with immediate relief. On the night of the 11th the expulsion of the tube was uneventful, recovery proceeding without further incident.

Case 10.—Seen with Dr. S., November 17, 1914, a boy, two years old, thought to be in extremis. The tube was placed on first attempt, within five minutes after arrival, with prompt relief. In this case the diagnosis was not made till after intu-

bation, the laboratory finding at first being reported as "suspicious." On the 23rd the tube was removed on second attempt at 8 a. m., to be replaced at 1 p. m., first attempt. On the 27th, removed at 8 a. m., replaced an hour later, one attempt sufficient for each procedure. December 2nd removed tube on third attempt, replaced an hour later on second attempt. During the manipulations on this occasion I recognized the probable cause of failure in my abortive efforts, which may also explain other failures. While the left index finger is holding up the epiglottis and palpating the laryngeal introitus, it monopolizes the operative field, and must be withdrawn sufficiently to give access to the laryngeal entrance, or further successful manipulation is prevented. But in this withdrawal care must be had not to release the epiglottis. That is what I seemed to have done, with resulting falling of the epiglottis, closure of the entrance, and passage of the tube into the esophagus. With the epiglottis down, no effort should avail to effect an entrance. Bearing this in mind, the next instrumentation in this case, an extraction, was without difficulty. On the 7th the tube was removed on first attempt at 8 a. m. For an hour the result seemed doubtful, but a chance was taken on leaving it out, and the case proceeded without further interference.

XX.

COMPLICATIONS IN TONSIL AND ADENOID WORK, WITH ESPECIAL REFERENCE TO CHRONIC SUPPURATIVE OTITIS MEDIA.*

By Harvey Mayer Becker, M. D.,

Sunbury, Penn.

The title of my paper, as it appears upon the program, is somewhat misleading, as it would indicate that I expected to deal with complications arising during tonsiloadenoidectomies, which is by no means my intention, and I trust you will pardon the attempt to discuss one complication present before operation.

I have a feeling of considerable hesitancy in appearing before you with this modest paper, and I hope you may not think that it is with any presumptuous idea of teaching you anything new, but rather that we may rehearse together some of those conditions which I desire to call complications in tonsil and adenoid work. I prefer to call them complications rather than sequelæ, inasmuch as they frequently antedate the attention to the tonsil and adenoid. We are frequently consulted for the purpose of treating the complications, and the cure of the complications depends upon the active and judicious management of the actual cause, of which the patient and his family are wholly ignorant, or have been grievously misinformed.

It will be obvious to you that it would be utterly impossible for me to take up for discussion more than one of the very many complications in the time allotted; nor would I feel myself justified or qualified in making the attempt. It should not be amiss, however, to name over some of them, just to refresh our minds and make us realize that there are other parts of the human body that should interest the laryngologist

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besides the much censured and abused tonsil, which we should well consider, in such a far-reaching combination of afflictions, before we proceed with our treatment, and which should be our aim and guide in determining the proper method of procedure.

We may say that a lymphadenitis of the globe and orbit is not rare, that we may have rhinitis, pharyngitis, laryngitis, accessory sinus disease, bronchitis, swollen and suppurating glands in the neck, obstruction to breathing, short lip, lack of development of the superior maxillæ, high palatal arch, deviated septum, irregular spacing and malposition of the teeth, which at once leads us into the comparatively new but most important field of orthodontia, defective speech, tuberculosis, nephritis, endo- and pericarditis, septic arthritis and metastatic abscesses, otitis, mastoiditis, perisinuitis, sinus thrombosis, brain abscess, and so on. These, then, are some of the conditions frequently present as a complicating factor in our tonsil and adenoid work; but there is only one which shall be considered at this time—otitis media. The mention of any complications during or subsequent to operative treatment has been intentionally omitted.

In the consideration of otitis, no attempt shall be made to describe the anatomy of the parts involved, except insofar as the anatomic structures may have a bearing upon the mechanical cause. It is quite certain that otitis, whether of a mild chronic variety, leading to tinnitus, otosclerosis and loss of hearing, or of a severer type, leading to an acute or chronic suppurative process, is produced by several modes, which may be said to be mechanical, including obstruction, inflammatory and bacterial. Several modes of mechanical cause may exhibit, as, for instance, direct obstruction by adenoid tissue in Rosenmüller's fossæ, or packed directly about the funnel-shaped orifice of the eustachian tube. Small adenoid vegetations so placed are capable of causing more harm to the ear than larger masses centrally located in the epipharyngeal space. Just what the exact relation of the pharyngeal musculature is to the ventilating processes of the ear is perhaps not any too well understood, but certain it is that the palatopharyngeus muscle extends to the pharyngeal orifice of the tube, so that inflammatory states in the closely associated tonsil, and necessarily in the pillars, may readily extend along the pharyngo-

palatin folds to the mucosa of the tube and thence to the middle ear. Persistent chronic and oft-recurring inflammatory conditions in the tonsil, and secondarily in the pillar and pharyngeal muscles, will greatly impair their function, resulting in the impairment of the muscular mechanism controlling the patency of the tube. Normally the tubal epithelium is ciliated, the movements of which cilia are toward the epipharynx, for reasons which are perfectly clear. From repeated inflammatory attacks in the tube this function is impaired or lost, and consequently when infecting material, laden with a variety of pathogenic bacteria, is forced into the tube by sneezing, coughing, blowing the nose, or by any of the numerous modes, the function of the ciliated epithelium being greatly inhibited or lost, these bacteria have an unimpeded passage to the middle ear cavity—a through ticket, so to speak—with rarely an attached return coupon. Once having gained access to the cavity, suitable media and conditions for prolific propagation having been found, it depends only on whether sufficient resistance to the invasion is present, whether or not a suppurative process is created.

A study of the bacteriology of suppurative otitis soon becomes convincing of the fact that a number of different germs are capable of producing this disease, the variety depending entirely upon the variety of germ present in the epipharynx at the time infection took place. Probably the most common is staphylococcic, which is followed by streptococcic as a close second. Pneumococci, the germ of influenza, whatever this may be, micrococci catarrhales, colon bacilli, and undoubtedly the organisms of scarlet fever and measles, which unfortunately have not been isolated with a certainty, but perhaps are closely allied to streptococci, are capable, either in pure culture or in mixed infections, of causing most intractable suppurations. Bacteriology has conclusively shown that any one of these or all of them may be present in the crypts, and even in the stroma of the tonsil which is diseased, from which they are easily distributed to the entire oral and nasal cavity. It is not the intention of the author to enter into a discussion as to whether they were bred there or whether they are being held there until their destruction could be accomplished—two much disputed theories at the present time, neither, perhaps, any too well founded on fact—but their presence there in large num-

bers, in increasing numbers, should be sufficient evidence to convict their host and shelterer as an accessory before the fact.

A diseased tonsil, therefore, whether enlarged or not, necessarily stands convicted of being capable of causing numerous otitic suppurations, which I believe to be incurable so long as this offending tonsil is permitted to remain in its fossa, and for this reason I begin my treatment of the otitis by a thorough cleaning of tonsillar fossa and epipharynx. The extracapsular operation in children, and the intracapsular in adults, are my choice. In adults it is frequently not possible to remove the capsule without injury to the pharyngeal aponeurosis, which is usually attached and adherent to the capsule from chronic inflammatory processes, and without exposure of the underlying musculature. I feel that it is wiser to confine the operation within the capsule, under these circumstances.

In this unhesitating complete removal of the tonsil I am not unmindful of its much disputed function, and most highly appreciate the endeavors of the many observers and investigators, the earnest pleas of such men as French and Jervey, for conservatism, and the untiring efforts of our esteemed G. Hudson Makuen in his constant endeavor to instill into our minds the fact that the unaltered throat and perfect voice are also important factors in the life and welfare of the individual, and a lack of quotation from their writings I would have construed as being due to limited time and not a failure in appreciation.

Certain it is, however, that if the offending tonsil has the marked effect upon the growth, hearing and mental development of children as just recently so well stated by D. Braden Kyle, from which there is no retraction, and certain it is, also, that when it has become sufficiently diseased to be the great factor in otitis, it has necessarily ceased functionating, just as any other organ does when severely diseased, and must be considered a menace, and of no further use to the patient.

Neither am I unmindful of the teaching that at the time of the tonsil and adenoid operation the ear had better be left alone, and subsequently treated on general lines for chronic suppurative otitis media. I shall say, at this point, that a tonsil operation should never be done during an attack of acute otitis.

I do not believe much can be gained in any discussion by the citation of individual cases, and shall not do so, but wish to present a series of twenty-two patients, with thirty-one discharging ears, discharging from two months to two years, in which treatment was given at the time of the tonsil and adenoid operation. These ears showed various germ infections, mostly staphylococcic and streptococcic, several with mixed infections, one of colon bacillus, and two postscarlatinal. Thirty-one ears are not a sufficient number to put the method upon a sound basis, but the results have been so universally satisfactory that I desire to present them to you.

Reasoning from the fact that infecting materials are more easily driven into the tube when perforation of the tympanum exists, the method appears to be contrary to the rule. I wish to say that the treatment has not been tried in acute suppurative otitis media without perforation, the tonsil operation having been deferred until the otitis has been cured, either by primary incision or spontaneously, when the ear probably will not need any further attention. To the present time it has been tried only on the chronic case with perforation present, and is based upon the principle that the whole air conducting mechanism of the ear has been infected, from the pharyngeal orifice of the eustachian tube to the meatus of the external auditory canal, and must be wholly asepticized at the same time.

METHOD.

Immediately after the tonsil operation the ear canal is wiped free of all discharge and the condition of the tympanum and middle ear cavity carefully determined as far as possible. If, as sometimes happens, the greater part of the tympanum has been lost, access to the tympanic end of the tube is considered sufficiently well established, but if only a small perforation is present, a long posterior incision is made, with an attempt to include the perforation in the incision. If this is not possible, owing to anterior location of the perforation, it is disregarded. After gentle aspiration with a Siegle otoscope, the canal is again dried. A five per cent alcoholic solution of iodin is freely placed into the canal, permitting it to flow through the perforation or incision into the middle ear cavity; and it is surprising with what ease it apparently finds its way down

the eustachian tube, by the favorable position of the head, not being retarded by air pressure. It may be found necessary to gently use the otoscope, alternately making pressure and suction. After a few moments a dry gauze pack is placed in direct contact with the drum, not too tightly, but sufficiently snug to prevent the passage of air upward through the tube and out through the canal. In about forty-eight hours the pack is removed.

Of the thirty-one ears thus operated, only four had not ceased to discharge at the time the pack was removed. One of these ceased after six days. The remaining three were the one colon bacillus and two scarlatinal. The colon bacillus ear ceased after four weeks; one scarlet fever after two months, while the other scarlet fever has not ceased after one and a half years, having been subject to several exacerbations during this time. In some of these cases granulation tissue was present in the middle ear cavity, which did not seem to retard the cessation of the otorrhea, but added considerably to the difficulty in properly filling the cavity with the solution. Small perforations closed very kindly under the stimulating effect of the iodin and pack, as did also the incisions. Several large perforations required some stimulating treatment afterwards, while a few very large ones I have not been able to close, even in the presence of what appears to be a dry ear. In the scarlet fever ear still discharging, the greater part of the tympanum was lost, with necrosis of the ossicles, and some more radical procedure will be necessary for this ear.

Details of the hearing present in these ears before and after operating would consume too much time, and for the sake of brevity these are omitted. As a class of suppurating ears in which usually considerable hearing had been lost, the great majority showed improvement that was very gratifying, while a few were not improved, notably one ear in which otorrhea had been present for over two years. In no ear was there less hearing after operating than before, another gratifying feature, in view of the fact that with the persistence of the otorrhea further loss would necessarily have taken place.

Naturally, one would inquire if the control of the otorrhea is to be permanent. The usual difficulty of following cases is

to be expected, but the explicit instruction to the parent, and so far as possible to the family physician, to return the child for inspection once a month, or at the first sign of any trouble with the ear, has usually met with success, and a fairly close watch has been kept over these cases, and none have been returned uncured except as above stated.

XXI.

THE IMPORTANCE OF THE VESTIBULAR MECHANISM IN NEUROLOGY.*

By J. Ramsay Hunt, M. D.,

New York.

The otic labyrinth is of great interest and important neurologically, as it is the terminal end organ of an extensive sensory mechanism which has connections with many portions of the central nervous system. This mechanism is frequently involved in affections of the nervous system—organic, functional and psychic—and when the seat of pathologic disturbances, produces symptoms which are essentially neurologic in character—e. g., vertigo—subjective and objective; disturbances of static consciousness and equilibrium; nystagmus, and forced movements due to an alteration of the labyrinthine tonus of the muscles of the trunk and extremities. A knowledge of the functions and relations of the vestibular nerve is, therefore, essential to a proper understanding of vertigo, equilibrium and orientation, nystagmus, and cerebellar localization.

ANATOMIC CONSIDERATIONS.

The otic labyrinth may be regarded as a highly specialized end organ of the vestibular division of the eighth cranial nerve. As is well known, the auditory nerve consists of two groups of fibers which are functionally quite separate and distinct—viz., the cochlear, which is the essential nerve of hearing; and the vestibular division, which is concerned with the important function of equilibrium and the space sense. These two groups of fibers course in a common trunk, the acoustic or eighth cranial nerve. Their peripheral and central terminations are, however, quite distinct and separate. In this discussion we are concerned only with the vestibular nerve and its relation

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to static consciousness and equilibrium. The peripheral stimuli which carry sensations of movements of the head take their origin from wave-like movements of the endolymph within the semicircular canals. The fluctuations in movement of these fluid columns are registered by waves of the delicate cilia on the epithelial cells of the cristæ ampullarum; from here the impulse is carried to the ganglion of Scarpa, a small peripheral ganglionic structure of the vestibular nerve and the homologue of the posterior spinal ganglia. From here the sensory impressions flow along the fibers of the vestibular nerve to the primary nuclei in the medulla oblongata. Of these there are several aggregations, of which the more important are the nucleus of Deiter and the nucleus triangularis; other accessory ganglia are the nucleus of Bechterew, nucleus of Staderini, and the nucleus vestibularis descendens. From these primary nuclei the labyrinthine impulses are diffused in many directions, viz.:

1. To the ocular nuclei on both sides, by way of the posterior longitudinal bundle. Through this mechanism vestibular nystagmus is produced.

2. To and from the vermis and cerebellar cortex, as well as other ganglionic structures in the cerebellum.

3. To the anterior horn cells of the spinal cord through the vestibulospinal tract. (The connections of two and three produce the forced movements, disequilibration and the alterations of muscle tonus of labyrinthin origin.)

4. To the region of the nuclear origin of the vagus nerve, which explains the nausea, vomiting, dyspnea, and circulatory changes of severe labyrinthine vertigo, and finally:

5. Fibers which pass cephalad through the midbrain and the optic thalamus to the cerebral cortex, terminating, it is supposed, in the region of the posterior portion of the parietal lobe. This communication with the cerebral cortex is the anatomic basis for the existence of static consciousness and affords an explanation for the disorientation and other psychic reactions of a severe vertiginous seizure.

Of great practical importance at the present time are the afferent and efferent communications between the primary vestibular centers in the medulla and the cerebellar cortex, which were demonstrated by Ramon y Cajal by the silver impregnation method. Cajal has also shown that collaterals pass from

the pyramidal tract fibers in the pons varolii to the cerebellar cortex of the opposite side. Therefore, the motor fibers of the pyramidal tracts stand in relation to the cerebellar cortex, and the labyrinth has extensive connections with the cerebellum and the spinal cord. These communications explain in part the occurrence of ataxia, forced movements, and alterations in muscle tonus which are observed in labyrinthine disturbances. They are also of especial importance in reaching a proper understanding of the reaction movements of the head, trunk, and extremities, as described by Bárány after the caloric test and rotation experiments.

This, then, represents, roughly, the course and terminations of the vestibular nerve, and is, so to speak, the anatomic basis of vertigo. It is a bilateral mechanism, and for its proper function a strictly harmonious action is required from each side simultaneously. If there is irritation or paralysis of the end organ on one side an asymmetric stimulation results, with the production of true labyrinthine vertigo, viz., sudden disturbance of equilibrium, nystagmus, a sense of subjective and objective rotation of objects, severe vertigo, nausea and vomiting, pallor, tachycardia, dyspnea, darkening of the field of vision, and sometimes unconsciousness.

LABYRINTHINE NYSTAGMUS.

The nystagmus of vestibular origin is rhythmic in character and consists of two components, a slow movement followed by a rapid return movement. The direction of the nystagmus —i. e., to the right or left, upward or downward—always refers to the direction of the rapid movement. It is always more manifest when the eyes are turned in the direction of the rapid nystagmus; it is diminished or suppressed in the direction of the slower component. The slow component is the direct result of the vestibular irritation, while the rapid return movement is of cerebral origin. Pike has shown, as a result of experimental study, that the rapid phase of the nystagmus is probably produced from the contralateral cerebral cortex. It is of especial interest to note that each semicircular canal evokes ocular movements in its respective plane.

Bárány has perfected an elaborate series of tests by caloric and rotation stimulation of the various semicircular canals, which are of great interest physiologically and of great prac-

tical importance. These tests not only determine the functional activity of the vestibular nerve, but a proper understanding of their detail and technic is essential for the interpretation of the various disturbances of labyrinthine tonus. Nystagmus of labyrinthine type is also produced by lesions of the vestibular nerve and of the central connections in the medulla. There is some doubt as to the relationship of cerebellar nystagmus to vestibular nystagmus. Wilson and Pike believe that pure cerebellar nystagmus is essentially an ataxic disturbance, an incoordination or asynergia of the ocular mechanism, and identical with the asynergic disturbances of cerebellar origin. While this may be true, it must not be forgotten that clinically most cerebellar lesions exert pressure on the underlying brain stem, and in this way may produce true vestibular nystagmus. It would also seem probable that irritation of the vestibulocerebellar fibers might have a similar effect.

MOTOR REACTIONS AFTER LABYRINTHINE STIMULATION.

That the labyrinth exerts a tonic influence on the voluntary muscular system was clearly shown in the epoch-making experiments of Ewald. The labyrinthine tonus is exercised through the connections existing between the vestibular mechanism, the cerebellum, and the spinal cord. It is homolateral —i. e., each hemisphere of the cerebellum stands in relation to the corresponding trunk and extremities. The movements of the body take place in the plane of the nystagmus and in a direction opposite to the rapid nystagmus—e. g., if with the head in the upright position and the right arm extended there is induced a horizontal nystagmus to the right, the arm deviates slowly to the left. Bárány has elaborated with great detail and exactness a series of tests for the determination of this tonus mechanism of the labyrinth and its influence on the movements of the extremities and trunk. This mechanism is homolateral in its influence—i. e., the right labyrinth and right cerebellum control the corresponding half of the body. As a result of these observations, he has reached the important conclusion that in the cerebellar cortex the various segments of the extremities are represented by four centers which are concerned with the direction of movements—viz., horizontally (right and left) and vertically (up and down.) A stimulation of the labyrinth on one side induces a corresponding alteration of

tonic innervation and the direction of movement. As a result there is produced in certain tests, what he terms pointing by. (Vorbeizeigen.) In other words, the sense of direction of movements in the trunk and the extremities is to some extent under the influence of labyrinthine stimuli, and the induction of nystagmus (rapid movement) in a certain direction produces a deviation of movement in the opposite direction. A very important phase of this question is its relation to cerebellar localization and the practical demonstration of cerebellar disease. It also supplements the experimental work on cerebellar localization of such observers as Rothman, Bork, and van Rynberk; and Bárány has shown that investigation along this line may contribute to a knowledge of the localization of centers in the cortex. For example, by freezing with ethyl chlorid, a portion of the cerebellar cortex which had been exposed by an operation, he was able to show that the posteroinferior surface of the cerebellum controlled the inward movements of the upper extremity.

VERTIGO.

It is important to recognize that vertigo, giddiness, and similar sensations are due to a disturbance of this large neural mechanism, and that clinically we should always think of vertigo in this concrete neurologic sense, just as in visual disturbances one thinks naturally of the peripheral organ of vision and its central connections. If this is done, much that is vague and uncertain in our clinical conception of vertigo will disappear. For example, in congenital affections of the vestibular apparatus, as in some types of deafmutism, the space sense is lost. These individuals may be rotated with impunity and yet experience no vertiginous sensation. What more practical demonstration could be offered to show the dependence of vertigo upon this mechanism?

Vertigo may result from labyrinthine disturbances, disease or injury of the vestibular nerve in the internal auditory canal and at the base of the brain, and affections of the central nervous system, more especially of the brain stem and cerebellum. Organic brain disease is one of the most important and most frequent causes of vertigo—e. g., tumor, abscess, multiple sclerosis, encephalitis, and the like. These may act directly

on the nuclear and tract connections of the vestibular system, but frequently the vertigo is produced as a result of secondary circulatory changes and alterations in the pressure of the cerebrospinal fluid.

The vertigo in functional conditions should also be referred to this neural mechanism; so that toxic, gastrointestinal, and neurasthenic vertigo represent limited functional disturbances, toxic or circulatory in nature, from sudden fluctuation in the vascularization of the vestibular mechanism. The not infrequent vertigo of cerebral arteriosclerosis probably has a similar orgin. As we have seen, the ocular mechanism is in close anatomic relationship with the primary vestibular nuclei in the medulla, so that ocular vertigo, especially in association with visual defects and diplopia, is relatively frequent. Ocular vertigo, however, is not purely vestibular, as vision itself is an important factor in static equilibrium and orientation.

A word in respect to psychic vertigo. We have seen that a distressing impression is one of the important accompaniments of a vertiginous seizure. It is an acute painful consciousness of disorientation, produced by disturbances of the vestibular apparatus, a disturbance of the judgment of the relation of body to space, a sensory deception as to posture, direction and distance—in brief, an hallucination of the body sense in its relation to space. We have all experienced vertigo in some of its forms, and retain a more or less vivid memory of the psychic impression; so that, especially in neurotics and in neurasthenic and hysteroneurasthenic states, psychic vertigo may become a dominating symptom. This may persist long after vertiginous seizures have ceased, as a phobia or obsession, and is a pure psychic disturbance, requiring psychic treatment for its relief. A rare and very severe form of this variety is that known as continuous vertigo, the vertigo permanens of Weir-Mitchell. Oppenheim has also described similar states as "Dauer Schwindel." This form of vertigo is clearly allied to the minor psychoses.

We should always bear in mind then that the higher cortical associations play an important rôle in vertigo, which in organic vertigo is the highly unpleasant realization of what is taking place, and in certain functional and psychic states is largely a mental image with associated fear and anxiety.

The treatment of vertigo in general need not be considered in a discussion of this sort. I would, however, emphasize the good results which have occasionally been obtained by lumbar puncture in cases of labyrinthine vertigo. This method was first suggested by Babinski, and Putnam and Blake in this country have reported favorable results. Bárány also recommends it; and from personal experience I can vouch for its occasional efficacy. From five to ten or even twenty cubic centimeters of cerebrospinal fluid are removed by lumbar puncture, with the usual precautions. It may be necessary to repeat the procedure once or twice at intervals of one or two weeks. The exact mechanism by which the favorable result is produced is still somewhat obscure. Probably, however, the changes in intracranial pressure, either circulatory or of the cerebrospinal fluid, is an important factor. It is also not impossible that the tension of the fluid in the semicircular canals may be relieved to some extent by this procedure.

XXII.

THE ROLE OF THE EYES IN EQUILIBRATION AND ORIENTATION.*

By Arnold Knapp, M. D.,

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Among the nervous impulses which act in preserving our equilibrium, those obtained from the eyes are mentioned as important. According to Gowers, the knowledge of the relation of external objects to the body gained from the contraction (i. e., innervation) of the eyeball muscles is one of the most important sources of guidance to the centers that regulate the muscular contractions for maintaining bodily equilibrium. Another important accessory to our sense of equilibrium is the static labyrinth. There is a close association between the eyes and the labyrinth. This association, however, is not directly connected with equilibration, but has a different purpose, as shown in the following:

The posterior longitudinal muscle which connects the various nuclei of the ocular muscles is in close relationship with the vestibular nerve. The vestibular nerve thereby is closely associated with the ocular muscles, just as it is with all the muscles of the body. It serves the purpose of maintaining the muscles in a state of tonic contraction. If this nerve be divided on one side, it will result in a complete loss of tone of the muscles of the body of the opposite side and in nystagmus. The vestibular apparatus is also in close relationship with the cerebellum, by means of Deiter's nucleus, so that the cerebellum is a further active factor in the regulation of our ocular movements. Bing calls it a reflex apparatus serving for the preservation of equilibrium, not only for the muscles of the body, but for the eyes. It receives centripetal impulses from the static labyrinth and from the ocular muscles by means of Deiter's nucleus, which assist in its regulating activity. On the other hand, it sends forth centrifugal impulses through this same nucleus, which

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preserve the position of equilibrium and control the ocular movements.

In certain animals the position of the eyes and their movements are governed nearly exclusively by the ear. The eyes in these animals, if the head is not moved, are practically motionless. As the head moves, the eyes make a compensatory movement in the opposite direction, which originates in the labyrinth. There is a constant action of the labyrinth on the ocular muscles. It holds the ocular muscles in check, giving them a definite tone, so that a lesion of one labyrinth causes a preponderance of the action of the opposite one. In man this association between the eyes and the labyrinth also works for producing compensatory movements, though these are not as important as in the lower animals. The purposes of these compensatory movements, according to Sherrington, are presumably for maintaining the normal relation of the eyes to the horizontal. These movements, Wilson and Pike have shown, are a direct reflex response, independent of the cerebellum, to insure that the retinal images shall not be disturbed by each motion of the head. It is well known that if both labyrinths are destroyed these compensatory movements are abolished.

The chief symptoms, so far as the eyes are concerned, which results from a lesion of the labyrinth, is nystagmus. Nystagmus, however, is a symptom which does not disturb our sense of equilibrium. In other words, those suffering from nystagmus do not complain of vertigo, unless of course, there are other agencies which affect the center of equilibrium.

Ocular vertigo is a very unusual symptom and is difficult to explain. Vertigo occurs, unquestionably, in ocular paralysis, owing to the confusion resulting from false projection; this, however, is a symptom which is surprisingly frequently absent. Muscular anomalies, such as insufficiencies of the ocular muscles, are practically never accountable for vertigo. It is stated by some that certain errors of refraction are apt to cause vertigo, particularly in those who are suffering from astigmatism with oblique axes. The term dizziness, as used by patients that consult an ophthalmologist, embraces many conditions and symptoms which have but little to do with vertigo; it is often impossible to analyze these symptoms or to explain their relation to an ocular defect, particularly when other factors which cause vertigo cannot be excluded.

XXIII.

A SUGGESTION REGARDING THE RINNE TEST.*

By Robert Sonnenschein, M. D.,

Chicago.

While various constructions have been placed upon, and different deductions drawn from, the test devised in 1855 by Dr. A. Rinne, we probably all agree with Bezold that of the tests usually made, namely, the Schwabach, Weber and Rinné, the latter is the most reliable. This is so because in applying it one can more easily get from the patient intelligent and distinct answers, thus noting more definite findings.

Rinne himself performed the test by placing the vibrating fork on the inner incisor teeth, but the method afterwards used by Bezold, Politzer, Urbantschitsch, and the other authorities (such as Boenninghaus, Gruber, Jacobson, etc.), is the one now in vogue, and probably used by all of us. This consists in striking the fork, the one advised by Bezold being a^1 (435 v. d.), and placing the stem on the mastoid process. When no longer heard, the prongs of the fork are held near the auditory meatus and the length of time noted by which the air conduction exceeds the bone conduction, when the test is positive. Where the Rinne proves to be negative, the air conduction is first determined and the fork then placed on the mastoid. Please pardon this trite statement in the presence of otologists, as it is made only for the sake of completeness.

It is not my intention now to discuss the various types of Rinne reaction, their clinical significance, nor their relation to the other functional tests, but will leave such consideration for another occasion. At present I desire merely to point out a fact, which, so far as my perusal of the literature goes, seems to have escaped the attention of most men of late, and that

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is the fact that the air conduction, as compared with that of bone, is really much greater than ordinarily assumed.

Bezold, in his textbook, considers that on the average the air conduction is about thirty seconds longer than bone in the normal positive Rinne, if the a^1 fork is used. However, in one of his great papers on the functional testing of the ear, he states that when the fork is held only at the meatus, without having been previously placed on the mastoid, the duration of air conduction is longer than when the usual Rinne test is done; in fact, it lasts seventy to eighty seconds. This is, of course, due to the fact that when the stem of the fork is pressed against the bone the excursions of the instrument are interfered with and the duration of vibration thereby shortened.

In another article he calls attention to the fact that when the Rinne test is carried out in the usual manner, and the fork no longer heard with the prongs held near the meatus, if the stem be then inserted into the auditory canal the fork is again heard via air, and usually for a period of about twelve seconds. This method I have used in only a few cases, and obtained similar results.

Since pressure of the stem against the meatal walls tends to diminish the duration of the fork's vibration, even this test does not show the full extent to which a fork may sound. Bezold claims that "we could really determine the length of hearing by air conduction only, if we were able to approach the drum membrane as closely with the prongs as we do with the stem of the fork."

Thus, while these facts are not new, it has seemed to me not wholly useless to consider them, first of all, because, as above stated, they seem to have escaped the attention of many, and, secondly, because it is often beneficial to test out the statements of others, with the idea of substantiating or refuting them.

Some have objected to the Rinne test as such, owing to the fact that by bone we determine the length of vibration of the stem, and via air that of the prongs, hence comparing the action of different structures. The answer to this is that the fork vibrates as a whole, and thus the stem and prongs sound equally long.

Then again, there is the objection raised to the method of testing by air alone, after having done the Rinne in the usual manner, on the ground that the fork would not each time be excited to the same extent. This error is obviated, it seems to me, by the technic employed. For quite a number of years I have in all tests (except where the very low forks are used) caused the fork to vibrate by a uniform method. In the thesis* read before this society some years ago, on "The Analysis of the Weber Test," opportunity was seized to emphasize the importance, at least from a scientific standpoint, of care and uniformity in doing the various tests.

To insure within reasonable limits equal application of force in exciting the a^1 fork, I always hold it at right angles to the body and allow a small pleximeter (armed with rubber pad) to fall of its own weight from a perpendicular position, striking the flat surface of the prong. By not using any force except that produced by gravity upon the head of the hammer, the personal element of variation in the impulse imparted to the fork is, I believe, almost entirely eliminated. If, indeed, there is a slight difference of a few seconds in the fork's vibration, this can have no material influence on the results. By this method, in testing the same individuals several times, as a matter of control, there is in intelligent persons rarely noted a difference of more than two to five seconds.

We can realize that many will contend that since the Rinne, as done in the usual way, gives us as good a ratio of the air to bone conduction as is needed for diagnosis, there is no necessity of further complicating the test by determining the air conduction alone after doing the regular test. However true this may be, it is equally certain that we do not in this manner really find out the actual or approximately actual duration of air conduction. But even if unnecessary for the ordinary practice of otology, is it not worth while to perform a procedure which at most requires one or two minutes extra in any case, and which may in time throw more light on the auditory function? At any rate, nothing can possibly be lost by doing this and recording the results.

These statements, of course, refer only to the positive Rinne, for in the negative one the full duration of air conduction has been found before the fork is placed on the mastoid.

*Read before the Chicago Laryngological Society, February 21, 1911.

In the attempt to see whether this difference between the air conduction, as shown after the fork, is no longer heard on the mastoid, and that noted by air conduction alone, twenty-five examination records of pathologic cases and twenty-five with normal ears were tabulated.

An analysis of these data shows that in the one hundred ears thus examined the excess of hearing by air alone over that shown in the regular Rinne averaged thirteen seconds. In the pathologic cases (fifty ears) the average was 10.7 seconds; in the normal cases (fifty ears) it was 15.2 seconds. The least difference was five seconds in a few instances, and the greatest was thirty seconds (Case No. 40).

The percentage of increased air conduction, when all cases were considered (one hundred ears), was twenty-eight per cent. In the pathologic cases the average was twenty-seven per cent, some being as low as eight per cent, and some as high as sixty-six per cent (Case No. 9).

In the normal cases the average found was twenty-nine per cent, some cases showing as low as fifteen per cent, and one as high as sixty-six per cent (Case No. 40).

It is interesting to note that there is thus very little difference between the reactions shown in the pathologic cases of this series at least, as compared with the normal ears. It is true that of the socalled pathologic cases the majority were tubal catarrh, or slight nerve degeneration. Perhaps the study of a series of marked nerve lesions would show different results.

It is rather striking that in many comparatively young individuals with negative ear findings the fork is often heard only a short time via mastoid—e. g., twelve to fifteen seconds; whereas, the majority heard via bone from eighteen to thirty seconds. This phase of the subject will be discussed more fully some other time. Where there is no nerve disease, its explanation may rest on various grounds, such as a difference in thickness of the bone, variation in pressure with which the fork is held against the mastoid, some peculiarity of the patient, etc.

It may seem strange to note in the list shown in the table of pathologic cases that so many of the ears designated as having a tubal catarrh show so well marked a positive Rinne.

While the findings of all the tuning fork tests could not be put down in the space at command, let me say that in those cases the high pitched forks were well heard, showing that no involvement, at least to any appreciable degree, was present in the nerve.

To summarize, it may be said that the duration of air conduction is considerably longer than is ordinarily noted in the usual Rinne test. While this excess is not materially different as between the normal and pathologic cases shown in our series, the examination of a large number of cases, particularly those with marked nerve degeneration, may possibly show a different ratio between the air conduction, as shown in the Rinne, and that determined without first placing the fork on the mastoid process.

In conclusion, let us suggest that the Rinne test be in every case carried out in the usual manner; then, if the reaction is positive, insert the stem of the fork in the meatus and note how much longer the sound is appreciated. Following this, excite the fork with the same intensity as the first time, hold it before the meatus only, and note the results by air conduction alone.

As before conceded, this consideration may prove to be more academic than practical in value, but why not apply a routine which requires but a trifling amount of extra time, and may in the end bring forth enlightening data?

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PATHOLOGIC CASES.

No.	Name	Age	Condition of ears	a^1 fork on mastoid		a^1 fork via air after no longer heard on mastoid		a^1 fork via air only		Increased duration of hearing via air, noted in seconds	
				right	left	right	left	right	left	right	left
1	T. S....	40	Tubal catarrh.....	20	30	65	60	70	70	5	10
2	E. J.....	33	Tubal catarrh.....	18	18	40	40	55	60	15	20
3	H. G...	56	Tubal catarrh and nerve degeneration	15	15	50	55	60	60	10	5
4	A. D. W.	33	Tubal catarrh and nerve degeneration	40	40	40	45	45	50	5	5
5	F. A. B.	65	Tubal catarrh and nerve degeneration	22	18	50	40	60	50	10	10
6	L. B....	23	Luetic nerve degeneration...	10	15	15	30	20	45	5	15
7	L. B....	60	Tubal catarrh and nerve degeneration	17	12	40	20	40	25	5	5
8	L. A....	29	Tubal catarrh.....	15	15	35	40	50	45	15	5

9	L. B....	19	Nerve degeneration.....	15	10	50	30	65	50	15	20
10	E. L. B.	19	Tubal catarrh.....	15	20	55	55	60	65	5	10
11	H. C....	38	Slight nerve degeneration....	15	10	45	40	55	55	10	15
12	A. E....	35	Slight nerve degeneration....	20	20	45	45	60	65	15	20
13	D. B. F..	69	Slight nerve degeneration and tubal catarrh	12	20	50	50	55	55	5	5
14	L. F....	16	Slight nerve degeneration and tubal catarrh	16	20	42	38	55	45	13	7
15	M. F....	21	Tubal catarrh.....	15	12	40	35	50	40	10	5
16	A. G....	42	Tubal catarrh.....	18	15	45	40	50	45	5	5
17	L. L....	21	Functional nerve degeneration	22	22	35	35	45	40	10	5
18	F. L....	47	Nerve degeneration.....	11	15	45	60	60	70	15	10
19	B. P....	22	Tubal catarrh.....	25	25	45	25	60	40	15	15
20	B. D. P.	6	Tubal catarrh.....	25	25	60	45	65	50	5	5
21	C. A. S.	52	Slight tubal catarrh.....	15	15	40	40	60	65	20	25
22	G. T. T.	25	Slight tubal catarrh.....	22	17	55	53	65	70	10	17
23	S. N....	54	Nerve degeneration.....	12	12	45	40	50	50	5	10
24	S. Y....	38	Tubal catarrh.....	22	20	40	30	60	40	20	10
25	E. F....	19	Otitis media chronica.....	15	20	25	45	35	55	10	10

NORMAL CASES.

No.	Name	Age	Condition of ears	a^1 fork on mastoid		a^1 fork via air after no longer heard on mastoid		a^1 fork via air only		Increased duration of hearing via air, noted in seconds	
				right	left	right	left	right	left	right	left
26	S. A....	19	Cerumen	20	20	45	40	60	50	15	10
27	A. B....	22	Cerumen	30	30	40	40	50	50	10	10
28	J. H....	50	Negative	20	18	62	50	75	65	13	15
29	C. G....	36	Negative	12	12	40	45	60	60	20	15
30	R. E....	26	Negative	15	15	45	50	55	60	10	10
31	E. S....	36	Negative	12	12	45	45	65	65	20	20
32	S. W....	30	Negative	18	24	40	50	60	60	20	10
33	S. H....	20	Negative	15	22	45	48	60	60	15	12
34	L. K....	37	Negative	12	15	45	45	60	60	15	15
35	W. F. L.	22	Negative	22	22	50	50	70	65	20	15
36	C. H. M.	26	Negative	20	28	55	50	65	60	10	10
37	S. T....	21	Negative	18	22	50	55	60	65	10	10
38	G. Y....	25	Negative	25	25	50	50	65	65	15	15
39	B. H. H.	23	Negative	18	18	40	55	60	65	20	10
40	J. V....	25	Negative	30	25	48	45	75	75	27	30
41	A. R. K.	25	Negative	20	20	60	60	75	80	15	20
42	R. W. C.	25	Negative	18	18	60	62	80	80	20	20
43	E. C. R.	23	Negative	25	25	60	60	80	80	20	20
44	L. A. S..	25	Negative	28	28	60	55	65	65	15	10
45	C. D. S.	24	Negative	25	25	50	50	60	60	10	10
46	G. G....	26	Negative	20	20	40	45	65	65	25	20
47	E. C....	25	Negative	20	20	55	55	70	70	15	15
48	S. R....	48	Negative	30	30	70	70	85	85	15	15
49	H. V....	25	Negative	25	25	60	60	75	75	15	15
50	H. S....	31	Negative	30	30	60	65	70	75	10	10

XXIV.

CHLOROFORM AS A GENERAL ANESTHETIC IN NASOPHARYNGEAL SURGERY.

By W. H. Nardin, M. D.,

Anderson, S. C.

Chloroform as a general anesthetic has for many years been held in great disfavor by physicians and surgeons in America, the belief that it is a dangerous drug in that capacity having so widely prevailed as to cause its use to be generally abandoned in the practice of surgery. Some states have taken so grave a view of its risks as to pass restrictive laws regarding chloroform. I must, in consequence of these facts, beg for these modest observations of mine an unprejudiced reading upon the part of those into whose hands they may fall.

We, as doctors, are often reminded that, despite the remarkable revelations of the past fifty years, medicine is not an exact science; hence the spirit of investigation often prompts us to go back and try again remedies, both medical and surgical, that have at some former time been rejected; and not infrequently the retesting gives us a better acquaintance with, and a clearer understanding of, these remedies in their uses as well as limitations.

Remembering that chloroform, although it has been discarded by most American surgeons, as a general anesthetic, is still conceded by all to be the best anesthetic in obstetric practice, despite the fact that a pregnant woman, particularly in the later months of pregnancy, is a poor surgical risk, I decided to give it a trial in my work, about six years ago. I have used it continuously since in all cases in that class of work where a general anesthetic was required, and out of approximately a thousand cases have not had one fatality, though some have come very near the borderline. But what operator does not encounter more or less trouble with an anesthetic when he

cannot have at all times an expert anesthetist, but is forced to use country practitioners with varied and, for the most part, poor training in the giving of anesthetics?

I have watched carefully the results of my work, and am convinced that chloroform, when properly given, is as safe as any other anesthetic for this class of work.

Furthermore, it possesses some advantages to which I would like to call attention. A very small amount of the drug will produce anesthesia, about two drams being sufficient for the operation for removing tonsils and adenoids, this being the maximum amount for the average case. The congestion is less about the head and neck, consequently less hemorrhage; there is less mucus in the throat, which is a great advantage in this class of work, since a clear field helps so much, both in the time consumed during the operation and in the ability to avoid injuring the surrounding tissues.

The shock is less, as indicated by the blood pressure, and its rapidity in recovering its standard.

The lowering of the blood pressure is less and is more fully gotten to normal, which is a distinct advantage in postoperative complications, as the normal resistance is not so long interfered with.

The stage of excitement does not last as long, nor is it so well marked as with other anesthetics; as a rule the patient goes to sleep as quietly as in his own bed.

It is much pleasanter to take, as there is less of the feeling of suffocation. This is very desirable with children, for they are as a rule not alarmed by it at the very beginning, and we all like, if possible, to avoid this condition.

It takes less time to anesthetize the patient, and, because of this fact, one accustomed to using ether will have to be especially watchful. In my practice the patient is usually well under in fifteen minutes, or sometimes in less time; that is, the corneal and pharyngeal reflexes have completely disappeared.

The duration of the anesthesia is not so extended, and the patient comes out more quickly. This is of especial advantage when the operation is performed in a private office, for, in a short time, approximately half an hour, the patient can be carried home. And I find that being carried into the fresh air, they more quickly recover; even if it is cold weather, if

they are well wrapped, there is no danger of bronchial complications, as when some other anesthetics are used. Patients sometimes go as far as fifty miles through the open country, the only inconvenience being the nausea and vomiting from the amount of blood swallowed during the operation. However, there is less nausea than usually follows any other anesthetic; nor does the nausea last as long.

There is less liability to postoperative complications, viz., bronchitis, nephritis, or pneumonia; for when the patient fully recovers from the immediate effects, one can feel that they are entirely safe.

Chloroform, since it can and must be given well diluted with air, requires no special or complicated apparatus, and can, therefore, be used in an emergency at any time or place by anybody; but to be of advantage to both patient and operator, it should be properly given in conformity to the following method:

First. Have an anesthetist who is in the habit of administering chloroform, one who will be directed during the course of the anesthesia, for most of the accidents from chloroform come from the one who goes to sleep on the job—and a careless anesthetist is more dangerous than the drug.

Second. Use only chloroform that you know to be chemically pure, and it must not be old.

Third. Always give it on an open mask, so that it will be well mixed with air.

Fourth. Give it drop by drop; never crowd it in the least; and the operator should never hurry the anesthetist, for the result may be disastrous to both the patient and the surgeon.

Fifth. The anesthetic should, when possible, be given in a room detached from the operating room, where the rattle of instruments an bustle preparatory to the operation will more than likely excite the patient, which we wish to avoid.

If these simple rules are carried out, it will be found that the drug is not so danger-fraught as we often suppose, but has many advantages of its own.

When the drug has not been taken well by the patients in my series, the following symptoms are noted: When I thought the patient should be fully anesthetized, there is some struggling, the pupils dilate, there is a sudden stoppage of respiration, slowing of the pulse, cyanosis, abolition of all reflexes.

The following treatment has been used effectually: Lower the head; open all windows, use artificial respiration, give hypodermic of strychnin, and adrenalin chlorid solution, and in some cases use oxygen gas.

SUMMARY.

For the removal of tonsils and adenoids, when a general anesthetic is necessary, chloroform is as safe as any other, and has much in its favor; that it must be given slowly and well diluted with air; that the danger comes from crowding it, by the operator or anesthetist being in too great a hurry; that the respiration stops before the heart, and that this stoppage comes on suddenly.

I write this with the hope that some observers more competent than I will test out my simple plan here suggested, and see whether or not we are neglecting a most valuable aid for lack of courage on our part.

"He who fights and runs away
Will live to fight some other day."

XXV.

A NEW MASTOID FLAP.

By Adair Dighton, F. R. C. S.,

Liverpool.

Having for years past struggled, with varying success, with the multitudinous flaps, described in the textbooks and journals, for the filling in of the postoperative mastoid cavity, it seems to me to be worth while to describe yet another, as it has the merit of simplicity, and in addition preserves the periosteum which, except in a few cases, has been entirely neglected.

The skin incision that I use is the classic one, extending from a point half an inch posterior to the temporal artery above and curving downwards about a quarter of an inch behind the attachment of the auricle, to finish at the center of the tip of the mastoid process. This incision goes through skin and subcutaneous tissue only, and during its performance the auricle is pulled strongly forward and dissected, almost peeled, from the underlying pericranium until the cartilaginous meatus is reached. The exposed pericranium is then incised by a cut extending from a point just behind the suprameatal spine upwards and backwards to a point just anterior to the line of the original skin incision. A second incision extends from a point just behind the lower angle of the cartilaginous meatus downwards and backwards, diverging from the first, to a point just within the line of the original skin incision. A third incision joins the posterior ends of these two, running in the same line as, but just anterior to, the original skin incision.

The pericranial flap, so formed, is raised from the bone and pushed forwards and held out of the way by the anterior plate blade of my self-retaining mastoid retractor, which is inserted between the pericranium and posterior osseous wall of the canal. The mastoid operation, whether Heath or radical, is then performed, and at its completion the retractor is removed

and the pericranial flap clipped and held with a pair of catch forceps. Next, a flap is made in the posterior wall of the cartilaginous meatus, after the method of Körner. The first incision is made along the junction of the posterior and superior walls of the canal and above the attached pericranium, whilst the second incision is made along the junction of the posterior and inferior walls of the canal and below the attached pericranium. The next step is to raise the pericranium along the posterior margin of the wound and, also, to separate it as far as possible from the overlying skin and subcutaneous tissue. This done, a catgut stitch is passed through the upper angle of the pericranial flap, and the two threads passed on a single needle through the upper angle of the raised posterior pericranium from within outwards. The same process is repeated with the lower angle of the flap to the lower angle of the raised posterior pericranium. The stitches are then pulled upon and the pericranial flap drawn under cover of the raised posterior pericranium and the stitches are then tied, the upper two threads to the lower two. In place of this catgut stitch a silkworm gut stitch may be passed through the pericranial flap from without inwards, and then from within outwards, and the two threads passed on a single needle under the raised pericranium and out through skin about half an inch behind the posterior edge, and tied tightly over a tube or roll of gauze.

Now, the Körner flap has been so cut, above and below the attachments of the pericranial flap to the cartilage of the meatus, that when the stitches, whether catgut or silkworm gut, are tied, the attached pericranium pulls on the tongue of the cartilaginous flap, draws it back into the postoperative cavity and holds it in position in identically the same way as it would be drawn back and held if the flap itself were stitched.

It has a distinct advantage in that it forms a pericranial covering to the cavity, and being in close proximity to healthy periosteum becomes united to it, and so hastens healing. The posterior skin incision is, of course, stitched in the usual way.

XXVI.

PAPER CLIP IN BRONCHUS SEVENTEEN YEARS, REMOVED BY SUPERIOR BRONCHOSCOPY.

By Edwin Beer, M. D.,

New York City.

During the past seventeen years numerous cases of foreign bodies removed from the bronchus by either the superior (laryngeal) or inferior (tracheal) route have been recorded. The following case is placed on record because of the long interval* between aspiration and removal, and to emphasize the fact that even after remaining so long in situ its removal was not difficult.

M. K., aged twenty-six years; admitted July 1, 1915; discharged July 19, 1915. History of present illness began seventeen years ago, when patient was playing with a paper clip which he had put into his mouth, and it suddenly became lodged far back in his mouth near the base of his tongue. His father, with the use of an ordinary teaspoon, made an attempt to remove the clip, but unfortunately pushed it still farther down the patient's throat. The patient said that he felt much better and did not notice any particular distress, and his parents believed that he had swallowed the clip, whereupon he was given castor oil. However, the next day, while the patient was playing in the street, he was suddenly seized with a paroxysm of coughing and raised a large quantity of mucus mixed with blood, also having great difficulty in breathing freely.

He was taken at once to a hospital, where he was placed under observation for a period of one week. After being put to bed his symptoms disappeared, and he was finally discharged without any attempt being made to recover the paper clip, of which he says he told the doctor.

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*Chevalier Jackson, Annals of Otology, Rhinology and Laryngology, June, 1913, reports a remarkable case in which he removed a collar button twenty-six years after aspiration.

After being home for a few days he was taken suddenly very ill, and his physician said he had pneumonia. He was sick at this time for about six weeks, having had what the doctor called a relapse, from which he finally made an uneventful recovery. He then enjoyed fairly good health for about two years, when he noticed a dull aching pain in the right side of his chest, and complained of a hacking cough with expectoration of large quantities of mucopurulent sputum, marked shortness of breath, and a sense of oppression and suffocation. Thinking that he was beginning to have some lung disease, he presented himself to his physician for examination. He was first told he had tuberculosis, and must come back for a second examination after a certain period, which he did, and was then told he had bronchial asthma. He received treatment for this last mentioned condition for a period of about twelve years in several institutions and from many physicians, but without relief. During this time he coughed more, sputum became very offensive and foul smelling, his pain and sense of suffocation increased, and he became exhausted on the slightest exertion, finally having to give up his work entirely. His weight, however, did not decrease with increase of his symptoms. His appetite was always good. Not being able to work, he gave up all hope and came to Bellevue Hospital, believing that he had tuberculosis, and applied for admission. Upon being questioned, he told of the paper clip he had had in his mouth seventeen years ago, and of the treatment he had received since that time. He was admitted to the hospital for X-ray examination on July 1, 1915.

Complaints on admission were:

1. Dull aching pain in right side of chest.
2. Paroxysms of prolonged coughing.
3. Expectoration of increasing quantities of foul, offensive, grayish sputum.
4. Shortness of breath, sense of oppression and suffocation.
5. Loss of strength.
6. Insisting that his trouble was due to the paper clip, which he said was in his lung.

X-ray examination showed a very distinct shadow of a flat-headed paper clip in the right main bronchus, with head down. It also showed marked thickening of the adjacent pulmonary tissue.

July 15, 1915. Superior bronchoscopy and removal of clip under combined ether and cocain anesthesia from right main bronchus. Cocain and adrenalin were applied in right bronchus to control reflexes, and ether insufflation (intratracheal) through metal catheter in the bronchoscope kept the patient thoroughly anesthetized. The clip was imbedded in the granulation tissue which almost filled the bronchial lumen, and there was copious discharge during the manipulations. As soon as one leg of the clip was seen it was firmly grasped with forceps and the clip withdrawn with bronchoscopic tube (Jackson-Rochester). In withdrawing the clip the two legs became spread apart, one entering the tube and the other trailing after.

July 17, 1915. There was very little febrile reaction. At first expectoration ceased almost entirely, and then it became more copious and then gradually diminished in amount.

July 19, 1915. Discharged feeling very well.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of December 16, 1915.

Orientation and Equilibration: A Study of the Sense of Position and Movement: Its Dependence on the Vestibular Apparatus and Its Importance in the Whole Field of Medicine.

DR. ARTHUR B. DUEL: After referring to the vast amount of literature that had accumulated with reference to our knowledge of position in space and adjustment to it, Dr. Duel spoke briefly of the work of Flourens, in 1828, who first demonstrated that the semicircular canals were definitely connected with the movements of the head; of Schrader's experiments on frogs; and of Professor Lee's experiments on sharks, which hardly left any doubt as to the participation of the semicircular canals and the labyrinth in the equilibration process. These and other physiologists had furnished proof that the vestibular apparatus is a special sense organ, solely concerned with orientation and equilibration; that its physiologic connection with the auditory apparatus is practically nothing compared with its intimate connection with other special senses, such as sight, the kinesthetic sense (muscle-joint), and the sense of touch. Hence, the main object of this paper was to bring out this fact, which was not generally appreciated, namely, that the eighth nerve, the auditory, should be described in anatomy and physiology as the nerve of hearing only, with its end organ, the cochlea; the socalled vestibular branch should be given a place by itself, and should be described as the nerve of orientation only, with its end organ, the vestibule and the semicircular canals. So far as the anatomic evidence in the matter was concerned, it might be observed that the distribution of the vestibular and auditory fibers was to entirely different centers. One had but to contemplate for a few moments the various precise poises of the

body in space, together with the adjustments of the limbs and other parts to the accurate localization required in daily existence, to be deeply impressed with the nicety of the mechanism which controlled them. The development of these adjustments to meet the demands of increasing activities was very rapid. A normal child of seven or eight years had unconsciously acquired a sense of orientation, by an adjustment of muscles and joints, which in magnitude far exceeded that acquired in later years by practice and training for the most difficult pursuits. This particular function of orientation was more important than any other in the struggle for existence. Its evolution might be traced from the stiffened tentacle of the jellyfish, through the calcareous particles of the higher forms to the more elaborate vestibular apparatus of the vertebrates, in which it became more important in all the acts of the individual. As the higher order of beings was reached, the sense of position was safeguarded and augmented by intimate association with other sensations, sight, the kinesthetic sense, hearing, touch, smell, etc. In the perfectly normal individual the conception of position was the result of the harmonious assembling of all these afferent impulses. Loss or imperfect action of any of them produced a disharmony which, if not corrected, resulted in a misconception varying with the degree of the upset. There might be simply a slight uneasiness or uncertainty at the mere thought of some daring act of equilibration, or some previous experience like looking from a dizzy height, a rough sea voyage, a ride on a merry-go-round, or other psychic illusory idea. Again, there might be a varying degree of vertigo resulting from the actual experiences, the thought of which might cause the psychic vertigo, and on to the more violent manifestations of vertigo accompanied by nausea, vomiting, loss of equilibrium, and ataxia. Through all this one should not lose sight of the fact that the vestibular apparatus was the most essential factor in the sense of orientation, the only source of such afferent impulses as had to do with that sense alone. The phylla deprived of its stiffened tentacle, the higher forms deprived of their otoliths, completely lost their sense of position and could not maintain existence. In the human, in whom the struggle for existence was the fiercest, it was quite natural that the compensatory factors for the loss of this sense of position furnished by the vestibular apparatus should be more

complex. A man deprived of one vestibular apparatus was temporarily upset in his sense of position; as a result he had violent vertigo, which his other senses made frantic efforts to correct. The violent effort to correct a change of position which was not actual, resulted in loss of equilibrium. These efforts to compensate for the loss of a vestibular apparatus succeeded with varying rapidity. The more violent manifestations subsided in a few days, and in a few weeks the ordinary acts were performed with apparent ease. However, in such a case the orientation could not be said to have become perfect again, but the individual had simply adjusted himself to the loss. Perfect orientation required angulation; impressions must come from the two different vestibular apparatuses at the same time, and the individual had learned the trick of moving his head or eye to get a point of view from two slightly different angles at practically the same instant. The deafmute born without vestibular apparatus, did many acts of equilibration with perfect ease and might miss many of the upsets of life which those with perfect orientation might have. He might never experience the throes of mal de mer. Nevertheless, the loss of both static labyrinths was a great catastrophe. While the first symptoms might not be violent, the task of learning to orient without the aid of organs specially designed for it was much like a blind person learning to read from the sense of touch, or the deaf one interpreting the movements of the lips from the sense of sight, and often the results were quite as imperfect.

To appreciate this one had only to try any of the more difficult acts of equilibration on such a patient. For instance he might be asked to try to stand on one leg, with the eyes closed, to walk forward or backward with the eyes closed, or an act of walking or turning in the dark. The normal individual might have the illusion when sitting in a train at a station that the train was moving, when, in reality, it was a train by his side that was coming in or going out of the station. This optical illusion was quickly corrected by the normal individual by looking at some upright portion of his own car which was not moving, or by closing his eyes, when the optical illusion was corrected by the static labyrinth. If a congenital deafmute were to have this illusion, it would have to be corrected by the sense of sight. If he closed his eyes the illusion would

remain, particularly if there were no vibration which would upset his tactile sense. The essayist stated that in having introduced this subject in a somewhat desultory manner he hoped he had succeeded in showing how orientation might be upset by misinformation from many sources; how impulses from the static labyrinth were so powerful to correct the misinformation, and, on the other hand, how misinformation from the static labyrinth itself was corrected with comparatively great difficulty by other afferent impulses. He would leave it to those who followed to point out the methods of fixing the cause of disequilibrium on peripheral or central organs, on functional or organic disturbance, on lesions of the different afferent sources of information, on the cerebellum, or the cerebrum, which, after all, must be the source of our consciousness of position as well as that from which we voluntarily changed it.

DISCUSSION.

DR. F. H. PIKE: I must confess to a certain hesitancy in speaking to you on anything apparently so well known as the otic labyrinth. The subject is so well known that the man in the street immediately associates the otic labyrinth and the cerebellum with the maintenance of equilibrium. And so firmly is the belief that the cerebellum is the great central mechanism for the maintenance of equilibrium fixed in the mind, that there is a strong tendency in some quarters to assert that the disturbances following lesions of the cerebellum and lesions of the labyrinth are essentially similar. But if we are to distinguish in clinical diagnosis between lesions of the labyrinth and lesions of the cerebellum, it appears to me that it is far more important to point out certain constant differences in the effects rather than to dwelling so strongly upon the resemblances.

Our knowledge of the effects of lesions of the labyrinth and of the cerebellum is derived partly from clinical observation and partly from experiments. We may point out that the difficulty of limiting the effects of a pathologic lesion to the area primarily involved is rather great. We may have, first, the effects of pressure resulting from an intracranial growth and also the increased secretion of cerebrospinal fluid which usually accompanies an intracranial tumor, and which will in its turn give rise to abnormally high pressures. This fact was an-

nounced from this platform about a year ago by Dr. Charles H. Frazier. In the case of intracranial or mastoid abscesses we have the spread of toxins and other effects upon remote parts of the nervous system. Secondly, there is the difficulty of getting human material for postmortem examination at the time when such examination would be most desirable for the pathologist.

The necessity for the experimental method of attack lies in the necessity of overcoming, in part at least, these difficulties. In the experimental method we may, if we so desire, avoid the effects of increased intracranial pressure and bacterial toxins upon other parts of the nervous system. Further, we may control the extent, both of the primary and of the associated experimental lesions, within certain limits. One must recognize, of course, that when a nerve fiber is separated from its cell of origin a degeneration will proceed to the furthest limits of the fiber, and to this degree the extent of the experimental lesion may be beyond our control. And finally, the experimenter may control the length of life of his animals, and get his material for postmortem examination at the time which he considers most opportune or advantageous. As an experimentalist I would, therefore, urge upon you the need for greater collaboration between clinical and laboratory workers. It is not my idea to supplant clinical observation, but to supplement it experimentally.

The lantern slides which were shown illustrated the effects of lesion of the labyrinth and of the cerebellum upon a considerable number of animals. These figures, for the most part, have been published in the Philosophical Transactions of the Royal Society of London, 1912, Series B, Vol. 203, pp. 127-160, and in the supplement to the Transactions of the American Otological Society, 1914, Vol. xiii, part II, by Prof. J. Gordon Wilson and myself. In all animals so far studied the effects of experimental lesions of the labyrinth result in a torsion of the head to the injured side. Immediately after the operation the animal may turn or roll over and over to the injured side. When both labyrinths are removed at once the animal—e. g., a dog—may refuse to stand unassisted, but lie with the limbs and head as closely applied to the floor as it can get them. The animal learns to stand after a few days, and to walk about without falling. But even months afterward it

will be unable to jump from even a moderate height to the floor without falling in a heap. This observation is originally due to Schiff, and we have repeated it many times. There are always certain deficiencies of this kind which may be discovered by careful examination of animals from which the otic labyrinths have been removed.

When one looks at the central nervous system from the point of view of its evolution, it is seen that in the lower forms of vertebrates the cerebral hemispheres form a very inconsiderable portion of the whole nervous system. But as successively higher animals are taken, we find that the anterior end of the nervous axis—that is, the cerebrum—shows a greater and greater degree of development. Experimental findings agree very closely with the anatomic development, and all the facts so far gathered point to the increased importance of the cerebrum in the daily life of the animal. And on the basis, both of the anatomic development and of the considerable number of experimental facts, it is our belief that one must look more and more to the cerebrum as the great central mechanism for orientation in space, rather than to the cerebellum.

Dr. Whitney said that it would have been difficult for the writer of the evening's paper to have selected a subject for presentation before this audience which would more thoroughly have tested his powers of analysis, his originality of observation, and his fairness of judgment. Nor could he, in his opinion, have offered a contribution more timely in its application to our present medical needs, or of greater practical importance to the entire medical profession.

Why is the subject chosen so difficult of practical treatment? Because it involves an explanation of complex conceptions and confusing manifestations which are of almost boundless variety and extent, and because it requires the reconciliation of numerous conflicting theories, many of which play an important rôle in any explanation which may be offered as a solution of the perplexing problems of equilibrium and orientation.

No elaborate argument is required to establish the indisputable value in the scheme of our daily life and activities of the function of equilibrium; it must, indeed, be regarded as second only in importance to those vital processes, respiration

and circulation, the interruption of which will speedily terminate the life of the individual.

How primarily essential to the existence of the animal organism is a knowledge of its position in space becomes the more readily appreciated if we permit ourselves a short excursion into the field of biology, the teachings of which science have clearly established that all forms of multicellar life present purposeful or somatic reactions which result in controlling their attitude toward or away from any source of irritation, in consequence of the possession of which faculty these organisms are able to select the most favorable position in which to place themselves.

In this appreciation of its relation to space, as indicated by recognition of restraint or freedom of motion, we may consider that we find our earliest suggestion of the function of orientation.

That an organism existing under primordial conditions should find some method, however simple, of determining its position in space essential to its survival, naturally suggests to the scientific mind a proposition which may be formulated in the following law: namely, in the progress of evolution through the succeeding higher grades of differentiation of structure, there must inevitably be found a development of this faculty (the sense of position) proportioned in its intricacy to the complex conditions governing the environment of this higher organism.

Such are in fact the logical steps whose orderly succession following through the lower organisms, thence upward to the human being with its highly specialized central nervous system, has resulted in our present conception of our position in space or orientation.

With increased complexity of structure and differentiation of the body form into head, trunk and extremities, there is developed a more accurate sense of balance, whether at rest or in motion, associated with a well defined sense of direction.

Thus, little by little, through an infinite series of gradations keeping pace with the physical needs requisite for the preservation of the animal, the sense of adjustment to surroundings is formulated, and under the stimulus of the will and intelligence at length fully established.

Intricate as are the physical sensations and nervous impulses, the correlation of which are required for the maintenance of equilibrium among the higher animals, they still are wanting in many of the complex conceptions which constitute essential factors in the function of orientation in man.

So remarkable are the functions attributed to the vestibular apparatus in its relation to equilibrium and orientation, as to suggest that this organ may properly be regarded as the guardian of our sixth special sense.

That the function of equilibrium is a congenital possession of the human being, there seems every reason to doubt; for it is common observation that the infant, although strong enough to stand firmly on its feet, and to jump vigorously when supported, cannot maintain its equilibrium unassisted until it has gradually mastered the difficulties of equilibrium.

In orienting himself a man readily finds his relation to an object that he can see by employing the aid of his visual powers, to an object that he can feel by the aid of his tactile sense, and to the direction and source of sound by the function of his auditory mechanism; numerous additional afferent sensory impressions, however, contribute to the establishment of effective orientation. Of the above mentioned factors, each bears an important relation to its fellow in the function of equilibration, and the derangement of any one element of the group is sufficient to disorganize the correlation of all; it is, however, in disorders of the vestibular apparatus that we encounter the most severe and sudden disturbances in equilibrium.

The infinite complexity of the afferent impulses, whose harmonious correlation is essential to orientation, have been clearly indicated by the writer of the evening's paper, and I shall not weary you with attempts at recapitulation, merely confining my further remarks to a brief comment upon those manifestations of disharmony with which, as clinicians and otologists, we are interested, and which ensue upon a disturbance of those components of equilibrium over which the vestibular apparatus presides.

The history of systematic and scientific investigation of the physiology of the inner ear, and especially of the complex function of equilibrium, dates back far over a century.

The questions involved are so intimately related to other chapters of physiology, notably of the visual perception, the sense of space, direction and position, and are so often suggested by the practical experience of the clinic and operating room, that observers in the most widely distributed fields have contributed to our present knowledge.

It is, however, only within the last decade that the widely diversified and oftentimes fantastic views entertained regarding the complex functions of the various organs concerned in establishment of equilibrium have received proper interpretation, and even at the present moment there remains an embarrassing array of incompatible theories and hypotheses, each with its enthusiastic advocates, which are absolutely irreconcilable upon the basis of established knowledge.

Whether we can accept the views that the stimulation on impulses in the static labyrinth are aroused by gravitational pressure, or are the result of the movement of columns of fluid (endolymph), is not essential clinically to our interpretation of the manifestations which ensue in consequence of irritation of the vestibular apparatus, however interesting such studies may prove to the student of physiology.

The writer of the paper has considered in its broadest physical application the significance of equilibrium and orientation as related to the vestibular apparatus, and some of his suggestions will, I anticipate, provide food for mature reflection for our neurologic colleagues.

Of such a nature is the perplexing problem presented by the deafmute, who with the destruction of both labyrinths develops a compensatory sense of equilibrium largely dependent upon visual impressions supplemented by the tactile and kinesthetic sense; his orientation, however, always remains defective. Such a person finds it impossible to maintain his sense of direction when swimming under water; moreover, owing to the bewilderment of his faculties from loss of orientation, a deafmute, although a capable swimmer on the surface, would be in danger of drowning from his own misdirected efforts, if he were suddenly plunged into deep water.

If one would appreciate in a slight degree the difficulties of orientation when undertaken without the aid of sight and hearing, let him close his eyes, hold his fingers in his ears, and attempt to walk straight ahead for any considerable distance.

He will begin his walk with assurance, lifting his feet for a few steps with his usual confidence, but before he has proceeded many paces he will begin to slide or push his feet along the floor, instead of lifting them, in order that he may preserve a better grasp on his position. His stride will shorten and his mode of progression gradually degenerates into a lumbering sort of shuffling of the feet, finally terminating in a loss of sense of position which brings him to a halt.

He experiences these difficulties, notwithstanding that he is assisted in his experiment by a normally functionating vestibular apparatus.

A person suffering with any structural vestibular changes would exhibit an exaggeration of these various manifestations, and would deviate toward the affected or weaker side, and with the application of von Stein's test, the disability would become progressively more apparent.

To the practitioner of medicine an enumeration of the complex array of motor and sensory phenomena, whose harmonious adjustment or correlation results in the function of orientation, is of interest chiefly as an exposition of a scientific demonstration; but he attaches far more importance to one practical suggestion which will enable him to treat intelligently the manifestations which result from the disturbance of this adjustment, and to which we have applied the name of vertigo, than to the elucidation of any theory, be it never so ingenious.

His vertiginous patient is usually a practical and insistent person, who demands assurance that his distressing symptoms will soon be relieved, and the doctor must have at his command an understanding of the condition which will enable him to decide whether the case is one of functional derangement which will soon right itself, or whether he is dealing with structural changes in the vestibular apparatus, nerve or cerebellum, from which he may apprehend a permanent disability or even a fatal termination.

Can we, then, indicate any definite diagnostic procedure which will clearly establish the causes of disturbances of equilibrium or vertigo in any given instance.

From the clinical standpoint of the otologist, we are accustomed to encounter vertigo in connection with labyrinthine

inflammations, meningitis, abscess or tumor of the cerebellum, and vestibular hemorrhage and hysteria.

In the first mentioned condition of infective labyrinthitis, except in the event of a metastatic inflammation (an exceedingly rare manifestation), the presence of suppuration of the ear would at once indicate the source of the disturbance, while the concomitant nystagmus and deafness would supply the needful corroborative evidence.

While in otitic meningitis the history of aural suppuration with rigidity of the neck and the presence of Kernig's or Babinski's signs, when supplemented by the information afforded by spinal puncture, will ordinarily establish the diagnosis.

With abscess of the cerebellum we are again guided by the presence of ear suppuration, but with cerebellar tumor there may be no evidence of ear disease. The vertigo associated with these lesions is usually attended by cerebellar ataxia, while the disturbances of balance, instead of diminishing in intensity with the duration of the disease, are disposed, in contradistinction to labyrinth inflammations, to increase in severity. Again, with abscess or neoplasm of the cerebellum, the direction in which the body falls is independent of the nystagmus if this is present, and does not change with altered position of the head; in addition to which evidence the "pointing-by" experiment should indicate a retrolabyrinthine affection.

Hemorrhage of the labyrinth is attended with complete loss of hearing and with vertigo of such violent character as to preclude all attempts at observing the reaction movements; however, the history of sudden deafness, with attendant vomiting, vertigo and characteristic vestibular nystagmus, will readily indicate the diagnosis.

With attacks of hysteric vertigo the middle ear on physical examination may show no pathologic changes, while nystagmus, if present, is not of the vestibular type, nor are the disturbances of equilibrium such as are caused by labyrinthine disease; the body may have a tendency to fall backward or forward or to either side—that is, it follows no definite rule—while in labyrinthitis the tendency is to fall always in the direction of the slow component of the nystagmus, whatever the position of the head. In hysteria, deafness may be simulated, but the static labyrinth will react to functional tests. (Braun-Friesner, p. 200.)

The study of the function of the inner ear and the logical systematizing of functional test is by no means complete, but enough has already been accomplished to show that we are progressing in the right direction.

No doubt, an elaboration and critical analysis of all rationally scientific methods will be of eventual service to the practitioner, and the final result of accurate and painstaking investigations in laboratory and clinic will bring theory and practice into much desired accord and correspondence.

The objections which have been raised to the errors inherent in this or that test, the exception to minor points of this or that theory, are details which are in nowise fundamental and cannot invalidate the principles involved, nor prevent their eventual application. In fact, the practical objections of competent clinicians and surgeons can serve only a beneficent purpose in correcting errors while they stimulate anew the search for truth. (Friedenberg, "Non-Acoustic Functions of the Labyrinth.")

Paper: The Role of the Eyes in Equilibration and Orientation.*

By Arnold Knapp, M. D.

Paper: The Importance of the Vestibular Mechanism in Neurology.†

By J. Ramsay Hunt, M. D.

DISCUSSION.

Dr. Frissell: The rôle of the general practitioner in discussing the subject of vertigo, especially after listening to the brilliant expositions from the special points of aurist, physiologist, ophthalmologist, and neurologist, is a trifle difficult. Why is he here at all? The answer seems to be that, as ancient Rome gave her successful general a triumph with captives at his chariot wheels, so our First Consul has consented to accentuate the papers of the evening by insisting on the violent contrast of an internist discussing the specialist's pet field.

The general man is, however, deeply concerned, in that to his inexperienced hand comes the patient seeking relief from real symptoms, and here comes both his perplexity and his

*See page 453. †See page 446.

duty—to which branch of the dazzling array of specialists should the patient be referred? Is it a case of labyrinthine vertigo, and is the aurist to be called upon; or is muscular weakness of the eye the predominant factor? Is it a cerebellar tumor or a disease of the central nervous system which demands the special attention of the neurologist? Or, more difficult still, is it one of those threshold cases on the borderland of epilepsy, simulating labyrinthine disease, so admirably described by Gowers?

Fortunately, the dissentions on the internal groups somewhat help the perplexed practitioner. Meniere's original description of auditory vertigo was of a symptom complex, of sudden vertigo associated with nausea, vomiting, and deafness on the affected side; in other words, a disease showing the same symptoms as our experimental animals in which the semicircular canals of one side have been extirpated, the reasons for which have been so admirably described by my predecessors.

But gradually it was found that all cases did not have either the classic symptoms of Meniere's disease, or the classic cause —destruction of the labyrinth—but varied in intensity from slight tinnitus with vertigo to the severe type originally described. So arose the classification into Meniere's disease and pseudo-Meniere's; the former having as its pathologic base the destruction of the labyrinth, while the latter was attributed to slighter forms of disease, such as otitis media, changes in pressure of the endolymph, cerumen in the external meatus, disturbances in the vestibular nerve in its course to the central nuclei, or even more remotely to tabetic changes and the functional explosive irritation of the higher centers in the epileptic aura and attack.

But if vertigo may be attributed to so many diverse sources in which the ear is affected directly or indirectly, what of those cases in which muscular incoordination in the eye produces diplopia and a false and uncorrected concept in the brain, the vertigo of multiple sclerosis—that psychic type caused by looking down from a height in one unaccustomed to mountaineering; the common incoordination of acute alcoholism, and that distressing form met with in "mal de mer"? Must the general practitioner be relegated to the undignified position of

"Mother Sill," and be altogether eliminated from the diagnosis and treatment of this common malady?

In its ultimate analysis, the causes of these symptoms are in many, if not in most, cases a distinctly medical affliction, as syphilis, arteriosclerosis, aortic insufficiency, the various types of anemias, diseases of the gastrointestinal tract, which are directly responsible, not only for the appearance of the symptoms, but in many cases for the pathologic base of the symptom complex, the latter forming only a localizing symptom of a constitutional disease.

One must conceive it to be the duty, then, of the general practitioner, in the presence of vertigo, by careful examination of his patient—including particularly blood, urine, blood pressure, and serologic examinations—to first determine the presence or absence of underlying constitutional disease; and, secondly, by careful examination of the eye, ear, and nervous system, to determine in which special field the immediate defect causing vertigo lies. If, as is probably the case, he is himself unable to apply the more complicated rotation and caloric tests in the ear, or make a thorough examination for muscular strain or defective vision in the eye, he should refer such a case to the better trained specialist for a more thorough examination.

But with his broader field of vision, he should be able to coordinate the examinations of his technically more skilled confréres, and will often be enabled to arrive at a more correct diagnosis in a difficult case.

In treatment, too, where a cure may not be obtained by a correction of muscle palsy, a regulation of pressure in the labyrinth or by operative procedure, his handling of the hypersensitive and anemic conditions may greatly improve, or in syphilis brilliantly cure, the underlying conditions.

Therefore, the internist's plea is that even in this field he may be allowed some slight recognition from his more skilled brethren, and may be able to retain his place in the care of so obscure and puzzling a condition.

Dr. Joseph Byrne said that a distinction should be made between equilibration and orientation. Equilibration is a means of orientation, but must not be considered as the equivalent of orientation. He was pleased to hear Dr. Pike say that we must look ultimately to the cerebrum for the mechanism

of disturbances of orientation following injury of the labyrinth.

Orientation, in general, may be said to mean the perception of one's relation to his environment. An old definition of truth is that it is conformity between thought and thing. Orientation may be defined as conformity between the immediate testimony of our senses anent our relations in space, and our conception of what those relations are or ought to be. We have, then, in orientation two fundamental elements: (1) the immediate testimony of our senses, and (2) our conception, based on education and experience, of what our relations in space under any given set of circumstances ought to be. It is evident that the second element is mainly psychologic, and forms the background for the interpretation and evaluation of the first element—i. e., of present incoming sensory impulses from all sources.

Insofar as element number one is in accord with element number two, insofar are we orientated. Conflict to a certain degree between these elements causes disorientation—i. e., uncertainty of one's relations, with consequent sense of insecurity, fear, etc. The labyrinth is no more the organ of orientation than many other sensory mechanisms—e. g., kinesthetic, visuoocular, tactual, etc.—which inform us immediately of the actual relations of our bodies in space. But information from these sources has to be integrated and evalued against the background of experience before they enter into the mechanism of orientation. Absence of element number two does not cause disorientation, but simply absence of orientation, or ignorance of one's relation in space—e. g., a child, before it has learned through the different senses its relations in space. The importance of element number two will be apparent from the consideration of a few examples.

When a person with eyes closed, seated upright in a swing, feet off the ground, is rotated about the long axis of the trunk in the direction of the hands of a watch, there is a primary horizontal nystagmus in the direction in which the subject is actually moving. If the swing be allowed to slow down gradually, there will come a time when the nystagmus becomes reversed (secondary nystagmus)—i. e., horizontal against the hands of the watch. At this moment, if the eyes be kept closed and the feet off the ground, the subject will believe his body has re-

versed its direction and is now turning against the watch—i. e., in the opposite direction to that in which it may be actually turning. If the feet be put on the ground the eyes still being closed, the subject at once is disillusioned. The conditions of this experiment are so unusual that there is little or no background against which to interpret the incoming sensory impressions. The only experience the normal individual has of impressions from violent horizontal nystagmus is that associated with active turning of his body about the long axis. In the experiment, this plus the impressions from the nystagmus constitutes element number two, and the testimony of all the remaining senses, under the circumstances, offers no conflict, hence there is no disorientation, but merely an illusion.

One more example: An individual, absorbed in thought, standing on a ferryboat before the latter is loosed from its moorings, allows his eyes to rest on the water streaming past. Presently he believes the boat is moving. Here the background consists largely of a state of expectation that his boat is about to move, and the testimony of the senses, so long as his eyes see only the moving water, offering no conflict, an illusion is begotten—viz., that the boat is moving. A new psychologic background is then formed, based on the illusory motion of the boat. Now, when the eyes turn from the moving water and rest on stationary objects—e. g., on shore—the testimony of the senses—i. e., element number one—comes into conflict, not with the testimony of the other senses, as so many authors claim, but with element number two, in which at that moment was a false conception—viz., that the boat was moving. A revulsion of feeling follows, coupled with uncertainty and insecurity, which we call disorientation.

Examples might be multiplied indefinitely, but enough has been said to show that the psychologic element or background is a fundamental factor in orientation, and that disorientation does not consist in conflict between the immediate testimony of the senses, but between the latter and element number two, or the background derived from education and experience.

Dr. Jacobi wished to make a contribution from his own experience, for digestion by the specialists. He himself is subject to one particular kind of vertigo, which he always experiences whenever he is on the second story of a building, looking out of a window for just a few seconds. There is very

little vertigo in his head, but a great deal in his feet and legs; they begin to tingle, and the feeling creeps up over the knees. A little cerebral vertigo is connected with the sensation. He would be very glad to have this explained, and explained soon, for he is believed to be an old man, and would like to be able to understand it soon. All his life he has been perfectly healthy, so far as he knows—he has had no brain disease, no tuberculosis, no syphilis—but just fifty years ago he had a typhus. It might be that he had something in his brain that gives him that peculiar vertigo or unsteadiness when he looks out of a window. He would like to know what it means, for nothing of the kind had been mentioned this evening, and it might be that in only this respect he was quite unique. He is sure he had it when he was thirty-five years of age. He does not remember having experienced it before his typhus, but he has had it ever since.

Dr. Duel thanked the men who had taken part in the discussion for their very able support of what he had felt to be a very desultory discussion upon a vast subject. The paper would have had but little value without the discussion.

The subject was one of much interest to otologists, and indeed to all whose attention had been directed to it. A few years ago Bárány had brought out certain functional tests whereby symptoms of an upset in the static state could be interpreted clinically with great exactness.

The psychic side of the subject was a most interesting field, and the discussion of it might well occupy an entire evening. No doubt, every one present had dreamed over it a great deal, but he himself did not wish to consume the time by adding any more dreams to the problem.

In talking with the President, Dr. James, over the best method of presenting this rather far-reaching topic before a general meeting of the Academy of Medicine, his first idea had been to present a series of cases which would illustrate the vertigo and other symptoms resulting from many different lesions, both peripheral and central; but he had found that in presenting the data it would be necessary to give in detail the results of a number of highly specialized functional tests, which even the specialists followed with great difficulty. For those who were not familiar with these tests an intelligible explanation would have required more time than was available.

Dr. James had, however, in the course of this preliminary talk, brought out a most interesting and ingenious phase of the subject which had not been mentioned during the discussion, and perhaps he would say a few words about it.

Dr. James spoke briefly of the result of posture upon the circulatory system.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of January 14, 1916.

Acute Labyrinthine Irritation Due to Traumatism (Faulty Paracentesis).

DR. J. H. GUENTZER: This report may not be of interest to the otologist, but is here put on record to emphasize a protest made by the writer on several occasions against the advice of some otologists to the general practitioner—i. e., to incise the drum membrane of every acutely inflamed middle ear, for the purpose of drainage and to forestall further pathologic involvement. This is good advice to the family doctor who is well acquainted with the minute anatomy of the middle ear, who has good illumination and a clear field; but it is dangerous advice to the practitioner who is not well grounded in middle ear anatomy and is poorly equipped for illumination. While some would hardly dignify a myringotomy as an operation, it is well to remember that carelessly executed it has led to fatalities. Hemorrhages from the wounding of adjacent blood vessels, injuries that resulted in permanent deafness, and the wounding and infecting of the labyrinth, resulting in meningitis and death, have been reported in this connection.

On May 31, 1915, the writer was called by the family physician to see M. K., a male, thirty-one years of age. The patient was lying in bed on his left side, and had been suffering from pain in the right ear for two days; no discharge. Since the previous evening he had been unable to walk.

Examination of the right ear showed the membrana tympani to be slightly reddened; no bulging; a pin-head perforation about the center of the superior posterior quadrant, with a droplet of serous red discharge exuding. The patient had a spontaneous horizontal nystagmus to the left and rotatory on looking up; on attempting to rise he fell toward the right. The hearing was not impaired; there was no mastoid tenderness; no temperature; pulse, eighty-eight. A diagnosis of labyrinthine irritation was made, but no assignable cause could

be elicited. Calomel and sodium bromid were recommended, rather as a placebo. This condition lasted ten days before the patient was able to leave his bed. At a subsequent office visit the patient confided the fact that his family physician was called the evening before the writer's first visit. The patient was told that he had an abscess in his ear, and the doctor used an instrument to open it, when immediately the patient became dizzy, nauseated, vomited, and was unable to walk.

The labyrinthine irritation was most likely due to disturbance of the footplate of the stapes. Fortunately, the middle ear and labyrinth were not infected, there was no discharge, and the patient escaped the dire consequences that might have followed such an infection.

Sigmoid Sinuitis With Abducens Paralysis in Acute Mastoiditis—Recovery Without Sinus Operation.

DR. J. H. GUENTZER: This case is of interest on account of its mild general course, with little evidence of sepsis in the temperature sequence, in spite of the fact that a sigmoid sinuitis was found on operation; and also on account of its abducens paralysis of the eye opposite to the involved mastoid without the usual clinical manifestations of sepsis, which rather substantiates the fact brought forward by Dr. W. H. Haskin, in his scholarly anatomic studies, that venous pressure on the sixth nerve involving its sympathetic fibers as it pierces the dura with the inferior petrosal sinus is responsible for the abducens paralysis. It is probable that an aseptic thrombus of the inferior petrosal sinus causes by pressure an edematous perineuritis of the sixth nerve, inhibiting its function. The absence of sepsis in this case seems to refute the opposing theory that such an ocular paralysis is caused by a toxemia.

The history in brief is as follows: On May 11, 1915, the writer called to see F. C., a boy, ten years old. The patient had pain in the right ear for one week, with no discharge. Examination showed the membrana tympani to be red and bulging, the mastoid quite tender, and the temperature 104°. A myringotomy was at once done under chloroform, given by Dr. Quinlan, the family physician. The patient ran a slight temperature, never exceeding 100°, during the next five days, with a moderate amount of pus discharge, but no abatement of the mastoid tenderness.

At the United Hospital, Port Chester, on May 16, 1915, a mastoidectomy was performed under ether. Pus and granulations were found in the mastoid cells and antrum. On removal of the necrotic area covering the sigmoid sinus, it was found to be of a decided yellow color, like a wax bean, and pulsating. The sinus was freely uncovered in both directions from the knee, and was found to be of the same abnormal appearance. A pus culture taken from the antrum at the time of operation was later reported by Dr. J. G. Dwyer as streptococcus mucosus capsulatus. Sinus exploration at this time was desisted from, partly on account of the fair general condition of the patient, as well as for the want of surgical assistance.

The urine examination was negative, and a blood count before operation gave a leucocytosis of 16,000, seventy per cent polynuclears, and twenty-nine per cent lymphocytes. Facilities for blood culture were not available. A convergent squint, diplopia, and a paralysis of the external rectus of the left eye came on the fifth day after operation. The ocular fundus was normal. Some anxiety was felt on account of the streptococcus mucosus capsulatus infection, but no further complications occurred.

Excepting for a rise in temperature to 103 3/5° on the seventh postoperative day, intestinal in origin, the case progressed in the usual manner, and the patient left the hospital after two weeks. After another month the entire mastoid wound was healed and the ear was dry. The left eye was again normal in three weeks.

DISCUSSION.

DR. FRIESNER asked if he was correct in understanding Dr. Guentzer to say that the hearing in the first case was unimpaired, and he wished to know whether any tests were made with the noise apparatus; he presumed that no functional tests of the static labyrinth were made during the height of the condition.

DR. LOUGHRAN did not think that one should consider he had made the hearing test properly until the sound apparatus had been used on both sides.

He then cited the case of a medical student who had tried to operate on his brother's ear, under local anesthesia. Just

as he was making the incision the patient jumped the wrong way, and the knife was forced into the cochlea, and his hearing was destroyed. This incident brought to the minds of the students the fact that a myringotomy is a major and not a minor operation.

DR. FRIESNER said that in Dr. Phillips' service at the Manhattan Eye, Ear and Throat Hospital there is a case very much like the one Dr. Guentzer had reported, excepting that no exploratory operation had been performed on the sinus. The patient was a young woman of twenty-two years. Dr. Phillips had performed a simple mastoid operation upon her left mastoid two weeks after an attack of acute otitis media. For ten days after the operation she ran an irregular temperature, with slow rises and falls, ranging between 100.5° to 102°. At the end of ten days, when he first saw her at the invitation of Dr. Phillips, she had some headache. She did not seem to be particularly sick, but her neck was a little rigid and the cervical vertebræ a little tender; beside that, she had an ankle clonus of the left leg. The fundus was normal. At this time she had a sharp rise of temperature to 104°, and Dr. Friesner did a lumbar puncture. The fluid was normal. Twenty-four hours after the lumbar puncture she had a temperature of 105°. Another lumbar puncture was then performed, for she was having severe headache and her neck was rigid. The fluid was again normal. A number of blood cultures were made and proved negative. Several blood counts showed 12,000 leucocytes, and seventy-five to seventy-six per cent of polynuclears. Dr. Phillips had almost determined to explore the sinus, when she suddenly became better; her headache disappeared as if by magic, and her temperature became almost normal; she now eats and sleeps well, and her neck is no longer rigid. Within the last three days, however, she has developed a sixth nerve paralysis on the left side. Of course, the diagnosis lies between a basilar process, a meningitis and a sinus thrombosis, or a phlebitis if not a thrombosis. The picture from the beginning, although she showed some of the symptoms of meningitis, was really not that of meningitis. She was not so sick as meningitis cases usually are, and the cerebrospinal fluid, of course, was negative. It would seem as though she had a phlebitis. In her present condition, however, Dr. Phillips' has concluded not to interfere.

DR. GUENTZER, answering Dr. Friesner's query, said that the case was seen in the country. He had been asked by the country physician to look over the patient, and had no opportunity to prepare to make any particular ear tests. His hearing was good with the usual tests of closing one ear at a time.

In the second case, the boy was never really very sick, but went through the entire illness with a very good appearance, and was fairly well, although he was combating a severe infection. He had these different signs and symptoms, and yet no high temperature or appearance of septic illness.

Complete Paralysis of the Cochlear Division of the Auditory Nerve, Associated With Normal Vestibular Reaction.

DR. HUGH B. BLACKWELL: A. G., a well developed and apparently healthy woman, twenty-seven years of age, was admitted to the clinic of Dr. Duel at the Manhattan Eye, Ear and Throat Hospital, on November 17, 1915, with the following history:

Six years ago the right ear commenced to discharge, and has been running ever since. This discharge has been unattended by any constitutional disturbance. Five years ago the patient suddenly lost her hearing in the left ear. This loss of hearing occurred during the night, while she was asleep, and was unattended by any dizziness, nausea, or difficulty in equilibration. Since this attack the hearing in the left ear has not improved, and the patient has been compelled to rely entirely upon the right or discharging ear for audition. She has borne three healthy children, and both she and her husband deny specific infection.

Aural Examination.—Right ear: Canal contains foul pus, large perforation in membrana tympani, granulation tissue in fundus. Left ear: Canal dry, membrana tympani intact, no signs of previous suppuration, normal luster. Inflation shows left tube to be open.

Functional Examination.—Right ear: Patient can hear a loud whisper and low voice. Left ear: With the Bárány apparatus in the right ear, patient is unable to hear the loudest noise. Weber referred to right ear.

The left, or deaf, ear was then irrigated with cold water. In thirty seconds, marked nystagmus with dizziness and sensation of nausea was produced on looking toward right. The

left ear after a half hour interval was irrigated with hot water, and the nystagmus was reversed, producing also dizziness and sensation of nausea. The duration of this after-nystagmus was about a minute and a half. The patient was then whirled in a revolving chair ten times in ten seconds, in alternate directions. After each whirling there was produced an after-nystagmus upon looking in the opposite direction from which she was turned, the duration of which was about thirty seconds.

On November 4th, four days after these tests were made, they were repeated, with substantially the same results.

On the day of the patient's admission to the clinic, she was sent to the laboratory and her blood was taken for a Wassermann examination; it was pronounced negative. She was then placed upon a provocative specific treatment of iodid and mercury protoiodid, and at the end of a week her blood was again taken for Wassermann examination and again pronounced negative.

Conclusions.—Some authors believe that the function of the vestibular nerve can be destroyed and at the same time the cochlear division of the eighth nerve remain unparalyzed. Theoretically, there is no reason why the reverse of this proposition should not occur also—namely, complete paralysis of the cochlear branch, the vestibular nerve escaping. However, in a cursory review of the literature, he had not been able to find any cases reported in which the cochlear had been totally paralyzed and the vestibular branch entirely escaping. The case is interesting in that it shows the pathologic independence of these two divisions of the eighth nerve.

DISCUSSION.

Dr. Dixon stated that he would not recommend the use of either potassium iodid or mercury as a provocative. Mercury will cause a negative Wassermann in some positive cases.

Dr. Braun said that it was a very unusual and interesting case. The sudden onset of the condition made it probable that it was due to a hemorrhage. Whether the hemorrhage occurred into the labyrinth or into the region of the nucleii of the cochlear nerve in the medulla is very hard to say. It is difficult to imagine a hemorrhage occurring into the cochlea

which would not also involve the vestibular structures. It is more probable that it was a small hemorrhage involving the nucleus or root of the cochlear nerve.

Dr. Guttman asked if Dr. Blackwell had examined the spinal fluid. This might have shown a positive reaction. It was quite a well known fact that the cochlear branch of the eighth nerve is more sensitive than the vestibular branch; the cochlear branch, therefore, may be destroyed, yet leave the vestibular branch unaffected. The reverse, however, is very rare—e. g., that the vestibular branch should become destroyed and leave the cochlear branch unaffected.

Dr. Dougherty said he wished to emphasize the fact that a negative Wassermann signified nothing either way.

Dr. Blackwell, in closing, said he was inclined to think that the lesion was in the peripheral apparatus rather than in the central nuclei. The patient had no dizziness on the following day, and no nausea. It was difficult for him to conceive of a central lesion occurring so suddenly without irritation of the vestibular nerve. Of course, it was impossible to secure salvarsan for provocative treatment last November. Suppurative diseases of the ear frequently attack slowly the semicircular canal system, and adhesions and walling off processes occur which theoretically would protect the cochlea, but in almost all of these cases the cochlea became affected.

Streptococcus Mucosus Capsulatus Infection of the Mastoid Bone.

Dr. Robert Loughran: Of the various pathogenic organisms producing purulent infections of the middle ear and mastoid process, the streptococcus mucosus capsulatus has come to be the one that most arouses interest, not only on account of its tendency to produce a very rapid general involvement of the entire bony structure of the mastoid, but also because of its tendency to develop "a most dangerous and insidious latent period which may justly occasion apprehension concerning the outcome of any inflammatory ear disease which has its origin in the activities of this infective agent."

In the opinion of many writers, it is a bacterium closely associated with pneumococcus, yet with sufficient characteristics individual to itself in its growth in various media, its reaction to immunologic and fixation experiments, and its pathologic reaction in diseased tissues, to entitle it to especial atten-

tion. All admit that much work still remains to be done in order to place it in the position of knowledge to which its virulence entitles it.

Buerger has divided the pneumococci into four groups—typical forms, small forms, large forms, bacillary forms—the third of which "may easily be mistaken for the streptococcus mucosus capsulatus, but are, as a rule, very definitely lancet-shaped, whereas the latter are possessed of a more rounded or biscuit-shaped form." His classification of streptococci is: (A) 1. Streptococci without capsules. 2. Streptococci with capsules. (B) Streptococci with mucoid capsules, which he describes as the streptococcus mucosus capsulatus, differentiating them carefully.

On the other hand, Hanes, in an "Immunologic Study of the Pneumococcus Mucosus," concludes that the organism described by Schotmuller "represents a well defined group with characteristics which indicate a close relationship to the pneumococci rather than to the streptococci"; and that the name "pneumococcus mucosus" should be adopted for this group, rather than "streptococcus mucosus." He further notes that in this series of cases of lobar pneumonia caused by the pneumococcus mucosus, sixty-six per cent had died, and that when the organism had been found in the blood, all had died.

Lyell concludes that the type reactions for the pneumococcus mucosus group are the same as for the true pneumococci; and it is interesting to note that the majority of the strains he studied (ten in all) were isolated from cases of acute mastoiditis.

Dochez and Avery present tables showing the relative occurrence of organisms of the different types during the year 1912-1913, and record the fact that in thirteen per cent of their cases the organism fell in the group classified as the pneumococcus mucosus, and that in 1913-1914, eight per cent were of the mucosus group.

While there is uncertainty among bacteriologists as to just where the organism should be placed in the classification of bacteria, there is no doubt of its virulence and insidious tendency for the production of late complications. For the purpose of recording the various symptoms and conditions which the streptococcus mucosus capsulatus may produce, three cases were cited at length, the reader of the paper concluding: that

in meeting this infection experience has taught that we are dealing with an organism whose power of rapid and extensive destruction of the mastoid bone may easily be able to combat the best efforts against it, and that in order to conserve the patient's best interests, early and frequent bacteriologic examinations should be made of all discharges in suppurative otitis media, in order to be in position to take advantage of every point that could be of value in determining the necessity for operative procedure; and, further, that having made an attempt to stop its destructive course by operation as soon as there is any indication of mastoid involvement, there is still an uncertainty of prognosis to consider, even after the wound is healed and the patient apparently is well.

DISCUSSION.

DR. DIXON said that the views expressed in Dr. Loughran's paper corresponded with his own ideas in regard to the streptococcus mucosus capsulatus. At the New York Eye and Ear Infirmary it was the routine practice to examine smears in every ear case, and his experience during the last ten or eleven years had led him to the conclusion that the mucosus capsulatus occurred in about five per cent of all cases. Since he first began to study this germ (a good many years ago) he has been impressed with its extreme virulence, and has acquired great respect for it. As Dr. Loughran had said, it is not possible to determine when the end has been reached. To illustrate the length of time which such a case will run, Dr. Dixon read the history of a case which he had reported in a paper read before the Medical Association of Greater New York in February, 1913, and which was published in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY of the following June. The total duration of this case was from May 24th to the 12th of December, when it terminated fatally. During this time the patient was under constant observation, and had four operations. Dr. Dixon said that of course this was an unusual case, but that they have many cases at the infirmary which go out apparently well, remain so for a month or six weeks, and then come back with meningitis. Two or three years ago Dr. Whiting had a private case, an adult male, who was in rather poor condition (leucocytes only 4200). He did fairly well and left the hospital not long after his mastoid

operation and returned to his home in Long Island, being dressed at Dr. Whiting's office for about a month; then on a Thursday he came in, and was progressing splendidly. On the following Saturday night he had a chill, was readmitted to the infirmary, and on Tuesday he died of meningitis. Such cases are not at all uncommon.

Dr. Dixon said he had been harping on this subject for a great many years. The "hurry up" case reported by Dr. Loughran was rather an unusual one, and he had not personally seen or known of such a case. It was a very rapid one. As a rule, these cases at the infirmary progress exactly as any other dangerous infection, but the trouble with them is that a point is reached where the germ apparently becomes latent, and there are no symptoms, except possibly a little sagging of the canal and the discharge. Some time ago he had been called in consultation by Dr. Adams to see a patient, sixty-three years of age, without temperature, pain or tenderness, and advised operation; but two advised against it. The patient asked if it would do any harm to wait a day, and Dr. Dixon replied that he thought not. The next day Dr. Adams operated and found dura exposed to the extent of three-eighths of an inch. The patient recovered. There may be no symptoms, no pain, no temperature—nothing but the discharge and the mucosus capsulatus. Cases of this nature demand at least an exploratory operation, if the duration has been over. two weeks, for the patient's life is in increasing danger with each succeeding day.

Dr. Page agreed with Dr. Dixon, that it was not safe, as a rule, to watch these smouldering cases with a streptococcus mucosus infection longer than two weeks. He had never regretted operating on such cases.

Dr. Guentzer said that some two years ago he had a streptococcus mucosus capsulatus case occurring in a little girl of eleven, and had reported it extensively in a discussion at the opening meeting of the section a year ago. The point that was particularly interesting was that the child had a mastoiditis in May, and went through the entire summer until late in September, and then died. She had a sinus thrombosis and jugular involvement of the right ear, and a metastasis two weeks later in the ankle joint, which went on to necrosis of the astragulus. A few weeks after that there was another metastasis in

the hip joint and femur, then in two of the ribs and shoulder blade, then in the hip joint of the other side, involving the pelvic bones. There was not a single spinous process of the back which escaped involvement; the skin and muscle from the occiput to the buttocks had sloughed away; the child also had an abducens paralysis of the opposite side. One of the men at the consultation said he had never seen a worse infection, and it was certainly the most typical one that he himself had ever seen.

DR. DIXON wished to know if any of the members present had seen or heard of a case of recovery from meningitis caused by streptococcus mucosus capsulatus.

DR. BRAUN said that he had seen a case, two or three years ago, with Dr. McCullagh, at the Beth David Hospital. He thought that Dr. McCullagh had reported the case at one of the section meetings. The patient was a girl, about twenty-five years of age, who had had a simple mastoid operation done. A week later she developed a hemiplegia of the opposite side, strabismus, rigidity of the neck, and a Kernig, and was very apathetic. She continually tore at her bandages, and was very hard to manage.

A lumbar puncture showed cloudy cerebrospinal fluid which contained large numbers of streptococcus mucosus capsulatus. It was deemed inadvisable to operate, as the case was thought hopeless. However, she improved, and in several weeks was entirely well.

DR. BLACKWELL said he had understood Dr. Loughran to state that he would operate on these cases at the first sign of mastoiditis. He could not agree with Dr. Loughran on that point. He had operated upon a number of these cases at the infirmary, and had seen a number of them get well without operation. One must remember in dealing with the capsulatus infection, as with all other infections, that there are two important factors to be considered, both of which are subject to wide variations in intensity: first, the resistance of the patient toward infection; second, the virulence of the infective agent. The variations in amount and intensity of these two factors have been responsible for the number of recoveries from mastoiditis caused by the capsulatus infection occurring in his own experience.

DR. HAYS asked whether any one knew of cases where the symptoms became less on account of the virulence of the organism becoming attenuated. Many of these cases seem to lose their acute symptoms and go on to a quiescent stage, and he had wondered whether it was because the germ loses its virulence, or whether it continues as strong as in the beginning.

DR. LOUGHRAN, in closing, said that the germ loses its virulence only in dry secretions, and that the virulence could be restored very promptly by putting it in a proper medium. Every man has to be guided mainly by his own experience. His own experience with this organism had made him very much afraid of it. He had seen one case of it in his own famiy, and could quote two other very interesting cases, one of them being in a small boy whom he had seen for Dr. McKernon, the other one occurring in a man who had an acute earache at six o'clock in the morning. Dr. McKernon saw him in the afternoon, and he then had an acute exudative otitis. The next morning the boy had a mastoiditis, and an appointment was made for operation that afternoon. The attending physician objected—that being his general attitude toward all surgical procedures. The boy was operated upon the second day, and had an immense cavity. He finally got well after eleven or twelve weeks. Had he been operated upon the first afternoon there would not have had to be such a large operation nor such a prolonged convalescence. In another case the patient was awakened at three o'clock in the morning with an earache. Early in the morning the drum ruptured. He began immediately to have signs of mastoiditis—a little fullness in the upper posterior portion of the drum and periosteitis in the canal, and suddenly developed a moderate mastoid tenderness.

New Instruments: An Improved Method for Draining the Tympanic Cavity in Purulent Otitis Media.*

BY JOHN GUTTMAN, M. D.

DISCUSSION.

DR. LOUGHRAN asked if there was complete healing of the drum in every instance. There is always danger of contraction

*See page 389.

later, with permanent perforation. It was interesting to know that Dr. Guttman makes a good sized hole.

Dr. Heller asked whether Dr. Guttman administered an anesthetic each time; for if not, he did not understand how the operation could be accomplished satisfactorily in every instance, especially with young children, for even a lancing causes a good deal of pain, and such a measure as that proposed requires even more time, and the patient would suffer more. He also took exception to Dr. Guttman's method of blowing out the eustachian tube with the catheter after opening the drum. That did not seem to be a wise procedure when the middle ear was filled with infected material. Such matter gets into the antrum readily enough without being forced there.

Dr. Hays said that Dr. Guttman had come to the infirmary one afternoon and operated upon about half a dozen cases by this method, most of them under local anesthesia. That in itself is almost as painful as opening the drum. So far as the puncture itself was concerned, the cases were all acute cases, and Dr. Guttman claims that the instrument is chiefly for use in chronic cases.

Dr. Hays said further that his chief objection to the instrument in its present form was that unless it was very sharp, one was apt to make the incision half or a third what was desired, and leave a little piece which would hang in and block up the opening. He had suggested to Dr. Guttman that interchangeable points be made so that one could always have a sharp one. In acute otitis where such excellent results are obtained with the knife, it hardly seems necessary to get out a special instrument; but in cases where the incision has a tendency to diminish in size or close very rapidly, it is more than worth while to make a good sized opening such as this instrument accomplishes.

Dr. Page said that in his experience he had seen little good result from repeated myringotomies. If after one, or at most two, free incisions the swelling and bulging of the drum membrane persisted, he had seen no marked change in its appearance affected by a third or fourth incision to promote better drainage. Usually the bulging in such cases was caused by swollen mucous membrane and granulations, and he thought a large percentage of them finally came to mastoid operation in spite of oft repeated myringotomies, whether performed

with a knife or trephine. Personally he did not feel that the trephine offered any great advantage over a knife of fair size.

DR. FRIESNER said that in applying this trephine to the drum membrane, the contents of the tympanic cavity can never be under such pressure as to offer sufficient resistance to cause the instrument to cut through the drum. Wherever the instrument is efficacious, the resistance necessary to cut or punch out the circle must come from the inner tympanic cavity, and there certainly must be some danger of injury, if it be only the mucoperiosteum, or possibly even the bulb. This problem had been put up to a mechanical genius a number of years ago, and he made the statement that no drum membrane, no matter how much fluid or pus the middle ear contained, would be under such tension that one could, by a circular punch or trephine, cut out the drum membrane clean without the resistance of the inner tympanic wall behind it.

DR. SCRUTON considered that there might be some difficulty in keeping the delicate trephine in good cutting order; he also believed a more or less prolonged experience in the use of the instrument would be required to develop an ability to manipulate it successfully in every instance. He stated that in his opinion the opening made by the trephine would give a free and uninterrupted drainage which cannot always be maintained by use of the myringotomy knife. The instrument presented by Dr. Guttman is worthy a trial by every otologist. It may prove a decided advance over the present myringotomy knife.

DR. GUTTMAN said he was very glad the instrument had brought out so much discussion. All he could say was that he had used it upon more than thirty cases, and none of them had showed an ill effect from it. Some of these cases had been destined to operation for mastoiditis by different surgeons, yet this operation having been performed and proper drainage established, the cases got well.

Replying to Dr. Loughran's query about a permanent opening: he had expected this objection, and for this purpose had brought two cases for observation, one of them being a colleague, operated upon a year and a half ago, who shows no sign of the operation in his drum membrane, and the other a case that had been operated upon more recently. In not a single instance did the opening remain permanently.

As regards anesthesia: he had used mostly local anesthesia,

applying three or four drops of a four per cent cocain solution in the upper wall of the canal. In some cases that worked marvelously, and the operation was absolutely painless; in other cases there was some pain. The operation had been performed even on some children of eight or nine years, under local anesthesia; they cried a little, but the operation took only a second or two longer than an ordinary paracentesis. A few cases had been operated upon under nitrous oxid, but in most instances, especially with adults, local anesthesia was employed, and five minutes later the operation was performed with hardly any pain in some instances and very little in others; as the operation requires so little time there was no real suffering in any case.

The paracentesis knife is surely quicker, as it is thrust in the drum membrane and the thing is done; but it may have to be repeated many times on account of the insufficient drainage. With this trephine one gets a drainage that could not be secured with any knife whatsoever. Most of the cases operated upon by this method had been acute cases, and the results were excellent. The colleague referred to was absolutely well in twelve days and had no more pain, and being a physician, he was well qualified to judge the result.

Dr. Friesner had claimed that there was not enough rigidity in the drum membrane and not enough resistance to secure a good opening. That was pure theory. Dr. Guttman said that he had performed thirty-odd cases, and got through the drum membrane in every instance with the greatest ease, and never had any bad results. Many acute and most subacute or chronic cases will get good drainage by this method.

Dr. Heller said that he had found local anesthesia very unsatisfactory in incising the drum membrane. Lately he had been employing somnoform, which gives a very short general anesthesia—lasting only a minute; and with a child it is really ideal. It takes half a minute to a minute to get the patient under the effects, and one has fully a minute for the work. There is no vomiting or other ill effects. Dr. Halstead had published an article in the *Laryngoscope* (August, 1915, p. 562), stating that he had had a great deal of experience with it and no bad results.

Dr. Page inquired whether somnoform is ordinary ethyl chlorid.

DR. HELLER said that somnoform is ordinary ethyl chlorid eighty-three per cent, ethyl bromid one per cent, and the rest is methyl chlorid sixteen per cent.

DR. LOUGHRAN asked if there was any danger point.

DR. HELLER replied that, practically, there was not. It is given until the patient is under its effect, and then stopped; a one cubic centimeter phial is enough for an ordinary anesthesia. One can determine the condition by the breathing. The patient is at first a little excited, and then goes off into a deep sleep. No assistance is required.

DR. GUTTMAN said that Dr. Heller had stated there was no danger to be feared from somnoform. It was invented by a French dentist, but there had been some fatal cases following its use, so one should not be too confident with it, for there is some danger connected with it. Nitrous oxid seems the safest and most sure anesthetic.

DR. GUENTZER said that he, too, wished to sound the same warning. Some ten years ago it had been tried at the Manhattan Eye, Ear and Throat Hospital for tonsil and adenoid cases, and a few of them did not do well. It is not so simple and safe as Dr. Heller seemed to think.

DR. HELLER replied that he, too, had tried it on tonsil and adenoid cases, and it was not at all satisfactory there. The dental supply man said that he knew of men who were using it, and came to the hospital to demonstrate it on two cases, and it was absolutely useless. It is not suitable for anything except very short operations. The effect is too evanescent for anything like a tonsil operation.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held November 16, 1915.

DR. GEORGE W. BOOT, THE PRESIDENT, IN THE CHAIR.

Case of Hypophyseal Disease.

DR. OTTO J. STEIN exhibited a patient upon whom he had operated almost a year ago by the transnasalsphenoidal route. The patient was in apparently good health up to four years ago, when she first noticed a reduction in vision in the left eye, which was soon followed by attacks of headache, becoming more severe and bitemporal in character. Associated with the headache was a vertigo, which later became so severe that there were four or five attacks daily, completely incapacitating the patient from any work whatsoever. After a year and a half the vision in the left eye began to rapidly decline, and then evidence of impairment in the right eye manifested itself. The patient came under the observation of Dr. George Suker about a year ago, and from a careful examination of her eyes, and with the skiagraphic findings (as could be readily seen from the plates shown), a diagnosis of hypophyseal involvement was made, and the patient presented to Dr. Stein for operation, which was performed about a year ago. At the time of operation the vision was practically nil, and she was obliged to have someone lead her about. This, with the severe headaches and vertigo, added materially to her suffering. The pupils were equal and reacted to light and accommodation. There was no extraocular muscular involvement. The left eye was totally blind, only slight light perception being present, the disc being choked, and evidence of neuritis present. The right eye showed a choked disc of three to four millimeters; no atrophy as yet, but there was a neuritis. No nystagmus.

The symptoms, from a pituitary gland standpoint, were as follows: Patient, thirty-three years of age; married; no children; menstruation began at thirteen and ceased at twenty-

three; adiposity—weight being two hundred and forty-five pounds; skin dry; scanty hair; polyuria; functionless genital organs, and some infantilism. There was no sugar in the urine. Wassermann negative. Blood pressure one hundred and twenty-five pounds systolate, ninety diastolate. Gastro-intestinal tract negative. No motor paralysis. No Rhomberg. Tendon reflexes slightly reduced. From these symptoms it can be readily seen that we had to deal with a case of pituitary adiposity, with a typical Fröhlich's syndrome.

The Hirsch technic was followed in operating—namely, a typical submucous resection of the posterior portion of the nasal septum was done, with the addition of removal of the right middle turbinate. This gave sufficient access to the sphenoid region. Great care is necessary in elevating the periosteum from the anterior face of the sphenoid, as it is essential to maintain an unbroken membrane throughout, in order to thoroughly protect one's retreat, and thus avoid the possible danger of infection of the meninges from the nose.

After entering the cranial cavity by removal of the floor of the sella, the gland readily presented, and after determining somewhat its character by exploring with a probe, and concluding that it most likely was an adenoma, he was satisfied to limit operative measures to a decompression.

After six days the patient made a rapid convalescence, and recovered quickly from the symptoms previously presented, and is today entirely free from headaches or vertigo, and vision has returned to normal in the right eye, and so far improved in the left that she performs all the duties of her household, including sewing, reading and writing, etc., without being conscious in the least of any deficiency in sight.

It is interesting to note, in this connection, that the rhinologist has at his hand a technic for performing an operation in this region that is so simple, and withal so satisfactory, and at the same time is free from the objections resulting from the methods employed by the general surgeon. On inspection of the patient's nose, one could never detect the slightest evidence of her having any former nasal operation, and the patient suffers no inconvenience from such a method of operation.

Case of Rhinophyma.

DR. STEIN said this case commenced in the same way that many such cases commence. This condition usually occurs in those who are exposed a great deal to the open air in all kinds of weather—that is, in engineers, firemen, conductors and motormen, drivers and teamsters. There is frequently an element of force back of it. This man noticed it after blowing his nose violently, when something seemed to give way. He felt a dilatation of the vessels. He had a slight rhinitis at the time, just as many of these cases have. After this he noticed the enlargement. Then the lobulated masses began to form. The nose feels soft.

In looking at this nose, one thinks of several conditions. One is likely, however, to think more of rhinoscleroma until you touch the nose. Then you notice the difference. This condition is nothing but a hyperplasia of the skin.

Probably the best exposition of rhinophyma has been given by Jacobi, of Freiburg, in his Atlas, but, like all dermatologists, he attributes all of them to some form of acne. This has been denied by rhinologists. In the case shown, there was no history of acne, and no evidence of it about the man now. Dermatologists claim that there is a hyperplasia of normal skin, but particularly of those parts in which there are sebaceous glands. The condition starts with a hyperemia, which disappears easily on pressure in the first stage. In the second stage there are enlarged vessels and veins, and, finally, these small masses of tissue, which they say is nothing but hyperplasia from the sebaceous glands, later forming lobulated masses, until they deform the nose and pull it down, as shown in this case. The third stage is much rarer than the other two, which are quite common.

Dr. Stein did not think this particular type has ever been recognized in women, which is another interesting point.

On looking inside the nose, it could be seen that the man was entirely free from any involvement of the interior of the nose.

Carcinoma of the Antrum.

DR. GEORGE W. BOOT presented a patient with carcinoma of the antrum. He lost his eye four years ago from an accident, and before he had any symptoms of cancer. He came

to Cook County Hospital three months ago, complaining of right-sided nasal obstruction, and wanted something done to relieve his breathing. There was a large polypoid mass present, which the speaker removed through the nose. The carcinomatous process evidently began in the antrum. A Denker operation was then planned to relieve the nasal obstruction, a radical operation being out of the question, since the growth extended across the median line in the roof of the mouth. When the external wall of the antrum was exposed, it was found destroyed to such an extent that a curette could easily be introduced. The interior of the antrum was thoroughly curetted out, the internal and external walls and much of the floor of the antrum being removed. The patient then left the hospital, but returned a day or two ago in the condition presented. The cheek is much thickened with the growth. The hard palate is much infiltrated, and the infiltration extends almost across the roof of the mouth. In the speaker's opinion, the growth is absolutely inoperable. He is receiving Coley's serum and is being treated by the X-ray, but with little hope of accomplishing any permanent good.

DISCUSSION ON DR. STEIN'S CASE.

DR. JOSEPH C. BECK, referring to the case of rhinophyma, recalled a case which he and Dr. Friedberg had seen, which was even more extensive than the one presented. He thought it might be interesting to tell what they did in this case in the way of treatment. The patient had a tumor that hung down over the mouth. He was a man about sixty-five years of age. He was operated, with excellent result. The operation consisted of a decortication of the whole area of the nose involved; then some skin from each side of the nose and face was brought together to cover the defect. It made a pinched nostril, but the result has been satisfactory, and there has been no recurrence. The growth was removed right down to the cartilage. Kaposi has reported this as necessary in these cases, so that no vascular and verrucous structure will remain.

Dr. Beck thought the result in Dr. Stein's hypophyseal case was excellent, and he, as well as the patient, was to be congratulated.

DR. STEIN, in closing the discussion, said that he intended to perform the decortication operation in the case of rhino-

phyma, as he understands that is the only method of treating these cases with any result. Dr. Beck said that in his case he took off all the skin down to the cartilage. Dr. Stein would think that a faulty thing to do; because you have to depend upon skin formation. You should leave a little layer of skin there for it to properly epidermize. If you take it all off, you get scar tissue contraction, unless you do a skin grafting, or draw the skin from the neighboring parts over the raw surface. If you are going to allow it to heal without any grafting whatsoever, it is a mistake to cut so deep. You have got to carve it out, as a sculptor would carve, with a razor, and dissect all around the cartilage, but always leave a little skin underneath for healing. With this method, he is led to believe that the results are very good.

DR. BECK said he was afraid he had been misunderstood. Unless you remove the growth down to the perichondrium, there is a recurrence, and the nose must be immediately covered. He did not wish it understood that he left the nose exposed or did any skin grafting whatsoever, but made a dissection from the side of the face, in the form of flaps, and brought them together over the tip of the nose, so that the nose was covered with a healthy skin. To carve out this tissue and leave some of the skin would prove futile, according to authorities.

Paper: Suspension Laryngoscopy, With Report and Presentation of Cases.*

BY JOSEPH C. BECK, M. D.

DISCUSSION.

DR. S. A. FRIEDBERG thought that certain points should be emphasized in connection with the use of the suspension apparatus. The first concerns that of technic. When the instrument is introduced the tooth-holder and spatula are very close together. This prevents satisfactory illumination of the lower part of the pharynx and epigottic region. If care is not used in manipulation, one may get an edema of the epiglottis and aryepiglottic folds; or if too long a spatula is used there is the possibility of it going back of the arytenoids into the mouth

*See page 330.

of the esophagus. Any unnecessary or excessive manipulation in the supraglottic region is very apt to cause an edema. The speaker has found this to be so, particularly in children with multiple papilloma of the larynx, in using the simple direct method in operation. This brings up the question that if we are to use this instrument in children, particularly in cases of multiple papillomata, whether or not it is advisable to do a preliminary tracheotomy.

Dr. Beck referred to the difficulty experienced in suspending some cases. This reminded the speaker of a case which both he and Dr. Beck had tried to suspend at the County Hospital a few days previously. In this case there was a diffuse papilloma, involving one cord, with aphonia of a number of months' duration. There was complete anesthesia, but it was impossible to keep the spatula in place on account of the gagging reflex. After several attempts they were obliged to desist. It was interesting to note that immediately following the suspension operation efforts the respirations went up to about thirty-six a minute. The patient was very dyspneic; pulse about one hundred and sixty; the girl could not breathe very well lying down, and had to sit up in bed. A sedative was given, and the next day she was in very fair condition. On looking into the larynx the next day some evidences of traumatism were to be seen. There was considerable swelling of the vocal cords, with whitish exudate over the false cord, showing that the tip of the spatula must have come up over the vocal cords, producing this traumatism. The larynx was also deeply injected.

Dr. J. Holinger said that Dr. Beck, in his paper, spoke of suspension laryngoscopy in patients suffering from asthma. Dr. Holinger wished to draw the essayist's attention to a series of articles on the treatment of asthma by means of this method. The attacks stopped at once, but the cocain was always given the credit and not the laryngostomy. The cocainization of the trachea, at the bifurcation, will stop even very bad cases of chronic bronchial asthma for one to many years. After that time, the same procedure may be repeated, with the same result. Patients who had gone through terrible trials with bronchial asthma have been kept in absolutely comfortable condition as long as the observation lasted.

Dr. Beck asked if only one application was necessary.

Dr. Holinger said yes—just one application at a time. If necessary, after years a second treatment could be given, but these cases are the exceptions. The exposition of the matter referred to could be found in the *Correspondenzblatt für Schweizer Aerzte*, two or three years back.

Dr. S. A. Friedberg wished to add one more point to his remarks, namely, in regard to the use of anesthesia in these cases. In any operation about the respiratory tract the use of prolonged anesthesia is dangerous. That has been found out especially by those doing bronchoscopic work. The speaker had one death on the table, while removing a foreign body, after about an hour. The anesthesia in this case was given by an expert. Jackson and others have recognized the possibly serious dangers in working about the respiratory tract, and Jackson rarely uses general anesthesia in children in doing foreign body work, and very seldom uses cocain. The speaker, also, in the last few years very seldom uses cocain. That is a danger that should also be recognized. In using an excessive amount of cocain we are apt to have a dangerous complication, as Dr. Ingals has pointed out.

Dr. Beck has asked the speaker why he does not employ this method in foreign body work. He has not felt the necessity, because it may be managed so easily the other way. At least, that has been his experience, and he believes Jackson inclines to the same view. One objection Dr. Beck himself brought out, namely: In the case of unruly children who are to be suspended, an anesthetic must be given in order to accomplish anything. In passing the bronchoscope, without the use of suspension, this is not necessary.

Dr. George W. Boot wanted to call attention to two points in the report. First, amputation of the epiglottis for tuberculosis or other conditions is very easy under suspension laryngoscopy. Second, in cases where edema of the larynx is feared following suspension laryngoscopy, it is advisable to do intubation as a precautionary measure. The speaker has seen papilloma of the larynx cured by wearing the intubation tube alone. It ought to be still more efficient after removal of the papilloma.

Dr. Beck, in closing the discussion, showed another case, in order to illustrate how well he could speak. Speech is the essential thing in these cases. When this patient consulted

him he was very hoarse. He had a fibroma on the cord, which was a good-sized growth, not as large as a pea, but just about that form. It dropped subglottically and would not come up readily. It looked like a little polyp. It was situated anteriorly. This growth was removed by the suspension method described.

Dr. Beck wished to make a strong plea, based upon his experience, for the use of this method for small growths, in accurate removal.

Dr. Boot spoke of intubation, and that is what the speaker had wished to say in answer to Dr. Friedberg's remarks. He has done that after removal of papillomas in children, so as to avoid the possibility of edema. He likes to use tracheotomy in papillomas in children for a longer period, because it prevents their recurrence, to some extent, and is considered good treatment.

The morphin is an excellent thing in these nervous patients, and hyoscin, which the speaker used, as well as scopolamin.

If Dr. Beck were to try suspension again on the patient referred to by Dr. Friedberg, he would give her morphin and atropin or hyoscin to get her quiet. He has found that this inability to suspend some cases is more of a mental condition. He did not see any exudates, but the fact that there was some trauma in this case should not deter anyone from the use of the method in other cases. The thing to watch out for is the trauma of the arytenoid, by too long a spatula. He was glad Dr. Friedberg brought out that point. We must thank Albrecht and Killian for using the extension on their spatulas for epiglottis. By using a little shorter spatula of the Lynch apparatus than the one exhibited and carefully avoiding the arytenoids, the operation is easier, and the view better.

Dr. Beck did not know about the articles spoken of by Dr. Holinger, namely, the treatment and cure of asthma by one application of cocain. That is worth while trying. He had a case at the present time of a man who has such severe attacks of asthma that it is pitiful to look at him in one. Adrenalin and cocain locally have never done him any good.

Dr. Holinger asked if he injected the cocain down to the bifurcation.

Dr. Beck answered that he did not.

Paper: A Suggestion Regarding the Rinne Test.*

By Robert Sonnenschein, M. D.

DISCUSSION.

Dr. Alfred Lewy wished to add a suggestion to those made by Dr. Sonnenschein in regard to the technic of the Rinné test, with reference to the reading of the results. He thinks the expressions "R. plus" and "R. minus" alone are most meaningless. A great deal of confusion has been caused by the use of expressions plus or minus, without indicating at the same time how much the air conduction and how much the bone conduction was relatively lengthened or shortened. We expect to find a minus or negative Rinné in a conduction deafness, but this is only relatively true. We find it in a severe conduction deafness, not in a mild one. On the other hand, it is very common to find a negative Rinné, but a bone and air conduction both shortened, but, nevertheless, minus Rinné, in severe cases of nerve deafness. The speaker believes that if we would indicate the result of our Rinné test by an equation showing the bone and the air conduction, and the normal for the fork used, a great deal of confusion would be eliminated from the literature.

Dr. George E. Shambaugh was pleased to listen to this discussion of the functional hearing tests, especially because this important part of otologic work is the one most frequently gone over carelessly. An experienced otologist is often able to make a tentative diagnosis with a good deal of accuracy from the history the patient gives. But a positive diagnosis of the type and degree of deafness can only be determined by carefully carried out functional tests. Taken alone, the Rinné test is much more valuable than either a Weber or the Schwabach test, but even the Rinné test cannot be relied upon by itself to make a diagnosis in all cases. It is important here, just as in much of our work, to have a regular method of procedure, and to run through a number of the more important functional tests in every case where the diagnosis is in doubt. A positive or a negative Rinné is often a relative affair, and it is only by studying this test in connection with the others that an accurate conclusion can be reached regard-

*See page 455.

ing the cause of the deafness. A shortened positive Rinné may have the same significance as a negative Rinné, and in advanced unilateral nerve deafness a negative Rinné on the affected side has the same significance as a positive Rinné does in most cases.

Dr. J. Holinger said that the international scheme for recording Rinné's test accepted that each part of the test should be noted separately. For example, strike the fork and note the bone conduction; then strike the fork again and note the air conduction; then draw the difference. This was done with the same idea that Dr. Sonnenschein brought out. But just there is where Bezold brings up the point that we do not need absolute numbers; we need only the difference of hearing by air and by bone conduction in the same ear for our diagnosis. To get that difference we have to strike the fork only once. Therefore, the question of hard or easy striking is avoided. If you have a certain amount of routine in the test, you will easily reach the point where you can make four or five tests in succession on the same ear, and not vary more than one or two seconds in your findings.

The argument of making original investigation may be accepted only conditionally. Dr. Shambaugh says that we have to stick to a certain routine for statistic purposes. The speaker would add that any time-consuming additions to this routine ought to be cautioned against.

Dr. George W. Boot thought functional testing of the ear is apt to be slighted. Several years ago he was in a clinic in Philadelphia where they had one tuning fork (laughter). In another clinic they had three or four forks and an old-fashioned Galton whistle. In one New York clinic they had a set of Bezold tuning forks, but did not use them.

In this connection he wished to call attention to the little book by Sonntag and Wolff on "Functionspruefung des Ohres." It not only gives the methods of making the tuning fork tests, but the tests of the vestibular system as well, and all in very compact form.

Dr. Sonnenschein, in closing the discussion, replying to Dr. Lewy and Dr. Shambaugh, said there are seven different kinds of Rinné, depending on the cases in which either the air or bone conduction are lengthened or shortened. As stated in the paper, he did not discuss this point, but simply desired

to call attention to the fact that the actual air conduction, simply from the scientific standpoint, as a matter of pure knowledge, is much longer than that ordinarily assumed by most writers. It is necessary to remember, as Dr. Shambaugh so well pointed out, in making any diagnosis, where using tuning forks—which are practically all subjective tests—that you are entirely dependent upon the statements of the patient. There is no objective method of measuring the acuity of hearing. It, therefore, is necessary to take a view of all the tests, as well as the other clinical data, in forming a diagnosis.

In answer to Dr. Holinger, he would say that he has on his cards the whole international formula, as decided upon at the Buda Pesth Congress in 1909, and he puts down the figures on these cards.

As far as stating that all you desire for the Rinné test is the difference between the bone conduction as determined on the mastoid, and then the air conduction, he agreed with Dr. Holinger, and stated that this ratio is essentially determined in the ordinary routine. It simply seems to him a desirable thing to know accurately how long by air conduction the tuning fork is heard in most cases. Has not our knowledge of hearing by air conduction taken on a different viewpoint since the introduction of the monochord? It was always assumed that high tones are best heard by air conduction, whereas, as a matter of fact, with the monochord it has been shown that by bone conduction the very highest tones are heard much better than by air conduction. Therefore, if we can find anything after a long series of observations which tells us anything more definite, it may be important in later on arriving at some conclusion regarding functional testing. So far as its requiring more time, the speaker was willing to grant that, but it took only a minute longer. That point he had emphasized in his paper. If there is anything in the method described, after examining several thousand cases, we will know it; at any rate, nothing can be lost by performing the test.

ABSTRACTS FROM CURRENT LITERATURE.

EAR.

The Streptococcus Mucosus in Its Prognostic Relation to Acute Suppurative Disease of the Middle Ear.

WINCKLER (*Archiv. für Ohrenheilkunde,* Vol. 96, p. 193). The consideration of this important subject from the clinical standpoint, which is so much at variance with the usually held views, deserves to be read in full. The author in the period from 1907 to 1912 saw only one case where the pus escaping through the paracentesis wound showed the streptococcus mucosus, as compared with fifteen cases of infection through the streptococcus pyogenes, and four with the streptococcus lanceolatus.

This case was a child of six years, where healing took place in eight days. There has never been any relapse. Four weeks after the attack the streptococcus mucosus was found in the tonsils when removed. Shortly before this a nine-year-old brother was seized with acute middle ear disease which showed streptococcus lanceolatus. This case required opening of the mastoid within twenty-four hours.

These experiences have caused the author to entirely give up the examination of the pus obtained by paracentesis as of no practical value, inasmuch as the child affected with the streptococcus mucosus made a prompt convalescence without mastoid involvement, while the brother, who showed streptococcus lanceolatus, immediately came to mastoid operation.

During the years 1907 to 1914 Winckler performed one hundred and nineteen operations for acute mastoiditis. In nineteen cases occurring in patients from sixteen to sixty-four years, streptococcus mucosus was found once in pure culture in a woman of sixty years, as compared with thirteen pure cultures of streptococcus, one of lanceolatus, one of staphylococcus pyogenes aureus, and two with staphylococcus albus. One patient suffering from infection with streptococcus albus and two with streptococcus pyogenes, the former forty-five years old and the latter fifty-eight and sixty-four years, showed

all the characteristic symptoms ascribed to a mucosus infection —absence of pain, absence of fever, characteristic appearance of the drum membrane, small quantity of serous secretion after the paracentesis, eight to ten days later healed middle ear; six to ten weeks later a relapse with complaint of headache and slight tenderness over the mastoid, which up to that time had been lacking.

The X-ray picture in all these cases at that time showed cloudiness of the bone, and the mastoid operations which followed revealed extensive disease extending into the cranial cavity with involvement of the dura. The older the patient the less variation there will be in all types of infection. A typical clinical picture with the streptococcus mucosus has no value, when found in a person of advanced years, inasmuch as other forms of infection can show the same picture.

As regards acute otitis in children, the author found that in fifty-seven streptococcus infections, twenty showed the streptococcus lanceolatus, five the staphylococcus pyogenes aureus, two the streptococcus albus, one the bacterium coli, thirteen mixed infections, and only in one, in a child of a year and a half, where there was a double-sided acute mastoiditis, was the mucosus found in pure culture.

Among the thirteen mixed infections there was one case of a child of three years, where the streptococcus pyogenes and mucosus were found together, and another case in a child of eleven years, a double-sided mastoiditis, where the streptococcus lanceolatus and the mucosus were found together. The case of pure mucosus infection showed a sudden onset with high fever, spontaneous perforation of both drums, rapidly increasing swelling, first behind one ear and then the other. A complete mastoid operation produced a prompt healing on one side. On the other side a radical was necessary. Both cases made prompt recovery, so that in four to five days they were discharged from the hospital.

As regards the rôle of the mucosus in mixed infection the author is in doubt. He is inclined to feel, however, that its presence is not of particular importance. The author's views in regard to the involvement of the mastoid in very severe acute otitis are at variance with those of most writers. They are based upon the study of the X-ray pictures in such cases. In many severe cases with profuse discharge the X-ray shows

the mastoid cells perfectly clear, not only at the beginning, but also at the end of the inflammation. In other cases at the very onset there is a decided differenec to be noted betwen the diseased and healthy mastoid. It is interesting to remark that so far he has not been able to note the disappearance of the cloudiness from the mastoid after the cure of the disease.

The importance of the X-ray in all acute cases is dwelt upon, although it is admitted that it is necessary that the mastoid should be pneumatic as well as the disease one sided, in order to permit of its use. Where the usual methods of examination fail in arriving at a definite diagnosis, much importance can be derived from a picture showing distinct cloudiness in the sinus region or one where it is shown extending into the pyramid. Such cases, in spite of an apparent favorable convalescence as regards the middle ear infection and of the sinus, of mastoid involvement, edema, pain, sensitiveness, etc., are to be regarded with caution. The cloudiness can indicate permanent changes in the mucous membrane or reparatory changes in the diseased bone. It can, also, however, mean an encapsulated focus of inflammation, deep seated, in the region of the sigmoid sinus, which requires only a new exciting cause to become active.

The examination of the bone removed from the patient of sixty years, suffering from pure mucosus infection, showed fibrous transformation of the medulla, containing chiefly lymphocytes with a few leucocytes; in other words, a chronic process with acute exacerbations. The histologic examination of the bone in the patient suffering from staphylococcus albus infection and with streptococcus infection, gave exactly the same picture. This was equally true of the microscopic findings in the bone of the child of a year and a half, with double sided mucosus infection, and of the case of mixed infection in the child of three years.

The prognosis in middle ear affection, where the involvement of the mastoid has been proven, always demands the bearing in mind of a possible complication. Where there is a favorable condition as regards the size of the middle ear and of the aditus, the infection can go on to complete healing. On the other hand, in many cases it is undoubtedly true that after years the foci of disease still remain, in spite of the fact that the disease in the middle ear has promptly healed. In such

cases one of two possibilities for further trouble is to be borne in mind. The diseased process advances in an insidious way from the foci that remain in the interior of the bone. Here they play an important rôle in the intracranial complications which develop. Indeed, they are of more importance than that usually ascribed to cholesteatoma. Second, the foci of disease which remain can become incapsulated and a healing produced or a quiescent state. Later the diseased area is lighted up by an acute cold or an inflammation of the tonsil, and any of the well known complications can develop.

Often in these cases the hearing is normal and the drum membrane intact, especially in young individuals. In children where the bone has been removed, evidences are found to show that the process was by no means an acute one, but rather pronouncedly chronic.

The importance of the embryonic tissue found in nurslings has not been definitely determined. The author agrees with Wittmarck that the middle ear inflammation may serve to prevent the transformation of the embryonic tissue in the early months of life, and in that way lead to middle ear and mastoid infection. He does not, however, agree with Wittmarck in his contention that in such infections of the mastoid in children there is little risk of complication except where the streptococcus mucosus is present. It is his feeling that there must be, in addition to the pneumatic form of the temporal bone, a wide-open middle ear and auditory canal, in order to insure a favorable outcome of the inflammation.

In all the cases of extensive disturbance of the mastoid where a pronounced pneumatization was present, he found a small antrum and narrowness of the middle ear. This is to be explained by the mechanic interference with drainage produced by the pronounced swelling of the mucous membrane in a small middle ear cavity. The infection of the streptococcus mucosus is then, as far as the author's cases are concerned, much less common than it would appear from the statistics of other operators. In one hundred and eight cases of tonsil enucleation the streptococcus mucosus was found only twice. Whether the infection with this organism has a characteristic peculiar to the region, or whether it can have only a temporary significance, he cannot determine. His statistics are to be compared with those of Mark, who found in his cases

of mastoiditis four and one-tenth per cent of streptococcus mucosus infection, as compared with Stütz, where it was present in thirteen per cent of the cases.

The pathologic institute at Bremen, where all the histologic examinations were made, is firmly of the opinion that the mucosus infection gives changes in no way different to that produced by other organisms. *Harris.*

The Influence of Vasomotor Disturbances in Childhood Upon the Organ of Hearing.

STEIN AND POLLAK (*Archiv. für Ohrenheilkunde*, Vol. 96, p. 216). After an exhaustive analysis of this subject the authors reach the following conclusions:

Vasomotor irritability in childhood gives rise, in addition to disturbances in various organs, to trouble with especial frequency in the organ of hearing. The clinical symptoms, as far as the ear is concerned, are the result, without question, of disturbances in the circulation in the organ of hearing, especially as the result of anemia in the blood vessels supplying the ear, particularly those in the region of the auditory nerve itself.

They are both of a subjective and objective character. The subjective symptoms are noises in the ear and painful sensations; the objective, fatigue symptoms involving the acousticus, the function of the cochlea, and in some cases of the vestibular apparatus.

As proof of the direct relation between the vasomotor disturbances elsewhere and those in the ear, are to be mentioned, first, the fact that evidences of disease involving the ear almost always show themselves in the symptom complex of the characteristic clinical picture of cerebrovasomotor disturbances (headache, vertigo, brain fatigue, etc.); and second, the fact that the effect on the function of the inner ear is always proportionate to the intensity of the vasomotor disturbances.

The question whether vasomotor disturbances can also give rise to organic changes in the auditory nerve can without hesitation be answered in the affirmative. In such cases all factors which can give rise to an abnormal function of the vasomotor nerves—infectious disease, physical and psychical trauma, disturbances of equilibrium—give rise to or at least contribute to the development of the disease in the inner ear.

In the treatment the shutting out of all factors which may injure the vasomotor systems, proper diet, careful hygiene, and especially control of all influences arising from the school, are of prime importance.

Especial emphasis is to be laid upon the fact that by the careful observation of disturbance in the organ of hearing as the result of vasomotor irritability, trouble in childhood can be recognized at an early age, the further development of which in later years must, under all circumstances, be feared. Attention at the proper time can check the development of the disease, or at least put a halt to its rapid development.

Harris.

The After-Treatment of Brain Abscess, With Special Reference to Abscess of the Cerebellum.

BUELLER (*Archiv. für Ohren-, Nahren- und Kahlkopfheilkunde,* Vol. 98, p. 58), in an exhaustive way, considers all the various controversial questions in regard to the immediate and remote treatment of brain abscess. He reviews the teachings of various operators, and then gives the conclusions of the procedure in the Erlangen clinic.

In regard to a large or small incision of the dura, he advises, on account of possible infection of the arytenoid space, an incision which is not too long. The temperature following the operation is apt to be elevated for the first few days, whereas the pulse frequency rapidly decreases. Vomiting is of frequent occurrence after the operation. Its significance often gives trouble. It may be due only to an irritation of the stomach, and on this account need not always be considered as an unfavorable symptom.

The changes in the eyegrounds are often to be observed for several weeks after the operation. Indeed, there may be an increase of the optic neuritis. They have no significance, however, from a prognostic standpoint. The nystagmus, as a rule, decreases after the opening of the abscess, but returns with increased force in case there is a retention to the discharge of the pus.

A particularly alarming symptom is the paralysis of breathing. This may occur several days after the operation. The explanation is to be found in increased brain pressure, although too tight a packing can be accountable for it. No way is

known to prevent this complication or to combat it when it occurs.

A prolapse of the brain is one of the most serious complications met with. It is dependent upon increased intracranial pressure and infection of the brain content. The most careful drainage, even by lumbar puncture when necessary, is essential to avoid this. Authorities do not agree whether a large or small incision is more conducive to brain prolapse. In the treatment, large incisions are of value. A broad opening conduces to much better drainage, and in that way the retention of secretion is avoided. For small prolapses a permanent bandage is sufficient. Large prolapses call for the removal of tissue where it is edematous and much infiltrated. The liability of a further prolapse after the incision demands careful consideration, however, before doing so. Great weight is to be laid upon the general treatment after the operation. All congestion of the head which may cause an increased blood pressure is to be avoided.

The position of the patient is also of importance. He is apt of himself to lie upon the diseased side, for especially in cerebellar abscess there are disturbances of equilibrium which give the patient a sensation of falling toward the diseased side. Neumann makes the patient lie on the side corresponding to the direction of the nystagmus.

In regard to when the first dressing should be done, the author advises, if there are no special indications demanding, that it be done before, and that it be changed on the second or third day. The frequency of the dressings depends upon the individual case. Care of the discharge from the wound is very important. Henke believes that in all uncomplicated brain abscesses which result fatally, the cause is to be found in faulty drainage. Adhesions of the walls from the fibrin deposited are not infrequent. These of themselves can cause retention of the infected material.

The author dwells upon the importance of bearing in mind the possibility of a second abscess to explain the persistence of the symptoms complained of. They are similar to those produced by the retention, and a differential diagnosis must be made between the two. The existence of multiple abscesses is denied by many authorities. Their etiology could be ascribed

to a metastatic origin; pyemia, especially, can cause their recurrence.

The choice between gauze packing and drainage tubes is gone into at length. Authorities in favor of each are quoted, Politzer, among others, making use in his clinic of iodoform gauze packing impregnated with perhydrol, five to fifty. In the Erlangen clinic a glass or rubber drainage tube is used when there is profuse suppuration, and gauze packing when the discharge is only slight. Iodoform gauze is generally employed.

The position of the tube is of importance, an unfavorable position leading usually to retention. When symptoms of retention arise, the permeability of the tube should be at once tested. Some authorities are in favor of removing the lining membrane of the abscess. Curetting of the abscess wall should be undertaken only in a most careful manner. The author leaves the wall entirely alone.

Boric acid powder and iodoform can be used in the abscess cavity upon occasion. If there is much odor due to anaerobic bacteria, a solution of peroxid of hydrogen can be used. Neumann is especially in favor of it.

For purposes of exploration of the abscess cavity, to determine its extent and direction, Ruttin and Neumann, among others, recommend digital examination. This is strongly condemned by other surgeons. Killian's nasal speculum has been used for this purpose. Mention is made of Whiting's encephaloscope for this purpose, but apparently the author has never used it. The Killian bronchoscope has been employed for the same purpose with success.

The routine use of irrigation is no longer to be recommended. In the proper cases and in the proper manner, however, it is not dangerous, and often of great value. In the Erlangen clinic it is regularly used. The all important point in irrigation is to employ as little force as possible. For this purpose a middle ear canula with a rubber ball is employed. The wound is then carefully dried and a fine boric acid powder blown in.

When in the treatment for symptoms of retention it is necessary to make a brain puncture, a number of instruments can be employed, the needle, scalpel, or forcep. None of these is without possible harm. Which is best to use depends upon

the particular case. The needle has the disadvantage over the knife, in that the pus mingled with the necrotic brain tissue can easily stop it up. The author uses first the canula. If this does not succeed, he employs a knife. The danger of an exploratory incision in the brain must be constantly borne in mind. Pathogenic bacteria can be easily introduced into the brain. The most scrupulous care and sterilization of all instruments employed is, therefore, necessary.

Encephalitis and meningitis are the two complications of brain abscess to be borne in mind. In more than fifty per cent of the fatal cases, meningitis was the contributing cause. The recognition of meningitis is often very difficult, on account of the necessity of differentiating from the symptoms due to the abscess. Only less dangerous than meningitis is encephalitis. It can follow directly after the operation, and be accountable for the paralysis of breathing.

The duration of the after-treatment is very variable. Politzer states from three to six weeks. In the author's three cases that resulted in a cure, the time was forty-five, fifty-eight and sixty-one days, respectively. The prognosis, in the author's opinion, is not to be regarded as altogether favorable. Indeed, even in cases reported as cured, Heimann feels that an interval of several years must elapse before this can be properly claimed, for often after a considerable interval complications and relapses take place. In a general way it may be stated that the prognosis depends upon the character of the complication, the size of the abscess, and, above all, upon the drainage conditions. One should be very careful in any case in making a prognosis. Death can arise from the abscess itself, if proper drainage is not secured and maintained, or it can come from the extension of the process into the brain.

The author concludes his paper by giving the histories of the six cases recently operated upon in the Erlangen clinic.

Harris.

LARYNX.

Thermotherapy in Certain Forms of Catarrhal and Tubercular Laryngitis. (Preliminary Report.)

MOURE AND GOT (*Revue de Laryngologie*, May 15, 1916). The method is that of Bier, applied to the larynx. It previously has been used in Germany in Killian's clinic by Albrecht, who made use of heated boxes.

Got has devised an electric heater so arranged that it can be connected with the street current. Further, it is so constructed that the temperature shall not mount beyond a certain degree, and so burns of the skin are avoided. A thermometer is provided for measuring the temperature of the skin.

The treatments are given preferably daily, and last from three-quarters of an hour to an hour.

The authors report their results in a series of thirty cases, and draw the following conclusions from their investigations:

1. It gives excellent results in cases of spasmodic laryngitis during treatment, but the benefit does not last beyond the conclusion of the treatment.

2. In acute cases of laryngitis thermotherapy causes a rapid disappearance, usually at the second, third or fourth treatment, of the troublesome symptoms, and diminishes to a considerable degree the length of the disease. In grip the cure is obtained less promptly than in simple forms of laryngitis.